In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 11-577V
Filed: May 24, 2019
To be Published

D.G.,	*	
	*	
Petitioner,	*	
	*	
v.	*	Influenza ("flu") vaccine; seizures;
	*	autoimmune autonomic neuropathy;
SECRETARY OF HEALTH	*	gastroparesis; POTS; myasthenia
AND HUMAN SERVICES,	*	gravis; conversion disorder
	*	
Respondent.	*	
	*	

Lisa A. Roquemore, Rancho Santa Maria, CA, for petitioner.
Justine E. Walters, Washington, DC, for respondent.

MILLMAN, Special Master

DISMISSAL DECISION[1]

On September 9, 2011, petitioner filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10-34 (2012), alleging that influenza ("flu") vaccine administered on August 23, 2009 caused her muscle weakness, fatigue, dizziness, excessive sweating especially after meals, whose onset was 10 days post-vaccination. On September 12, 2009, she alleged she had seizure and fainting after breakfast. On September 17, 2009, she alleged

[1] Vaccine Rule 18(b) states that all decisions of the special masters will be made available to the public unless they contain trade secrets or commercial or financial information that is privileged and confidential, or medical or similar information whose disclosure would constitute a clearly unwarranted invasion of privacy. **This means the decision will be available to anyone with access to the Internet.** When such a decision is filed, petitioner has 14 days to identify and move to redact such information prior to the document's disclosure. If the special master, upon review, agrees that the identified material fits within the banned categories listed above, the special master shall redact such material from public access. On August 16, 2017, petitioner filed a status report in which she informally moved to change the caption to reflect just her initials "for privacy reasons." S.R., at 2. On the same date, the undersigned granted petitioner's informal motion to redact her name to her initials and ordered the Clerk of Court to change the case caption, which the Clerk of Court did. This case is already redacted.

vasovagal syncope,[2] benign systolic murmur, positive ANA,[3] multiple somatic complaints, anxiety, dystonia, neurocardiogenic syncope,[4] and postural orthostatic tachycardia syndrome ("POTS").[5] Pet. at ¶¶ 9, 12, 13, 15, 15, 19, 20, 23, 25, 28. 31, 34, 38, 41. Petitioner's affidavit, dated September 7, 2011, was attached without an exhibit number.

PROCEDURAL HISTORY

On September 9, 2011, this case was assigned to former Special Master Daria J. Zane.

On February 21, 2012, petitioner filed a status report regarding record collection in which petitioner's original counsel Robert J. Krakow stated that he was having difficulty obtaining treatment records from Dr. Rashid A. Buttar because of an unpaid bill reflected in Exhibit 56. S.R., at 5. Mr. Krakow states that Generation Rescue offered to pay for all medical treatment and expenses for petitioner's medical treatment by Dr. Buttar, an osteopath. Id. at 6. The condition for Generation Rescue's payment of petitioner's medical treatment and expenses was petitioner's agreement to being on video during Dr. Buttar's treatment of her. Id. Mr. Krakow states that for reasons Generation Rescue did not explain to petitioner, Generation Rescue discontinued its involvement with Dr. Buttar's treatment of petitioner and refused to pay her medical bills for that treatment. Id.

On May 9, 2012, petitioner filed as Exhibit 58 an external terabyte hard drive with the name "DG Copy" consisting of files of videos. Each video is marked by the name of the digital file or folder as the files appear on the hard drive.

On July 16, 2012, petitioner's counsel filed a motion to withdraw. Mot. Petitioner wrote her counsel on May 30, 2012, stating that "she wished to 'take over representation of my case' and instructed counsel to discontinue representation of her before the Court for all purposes." Mot., at 1.

On October 2, 2012, petitioner filed a consented Motion to Substitute Attorney, which former Special Master Zane granted on October 22, 2012.

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[2] Vasovagal syncope is "a transient vascular and neurogenic reaction marked by pallor, nausea, sweating, bradycardia, and rapid fall in arterial blood pressure which, when below a critical level, results in loss of consciousness and characteristic electroencephalographic changes. It is most often evoked by emotional stress associated with fear or pain." Dorland's Illustrated Medical Dictionary 1818 (32nd ed. 2012) [hereinafter, "Dorland's"].

[3] ANA or antinuclear antibodies are "antibodies directed against nuclear antigens; ones against a variety of different antigens are almost invariably found in systemic lupus erythematosus and are frequently found in rheumatoid arthritis, scleroderma (systemic sclerosis), Sjögren syndrome, and mixed connective tissue disease. Antinuclear antibodies may be detected by immunofluorescent staining. Serologic tests are also used to determine antibody titers against specific antigens." Dorland's at 101.

[4] Neurocardiogenic syncope is "a serious type of vasovagal syncope precipitated by a stimulus that causes either bradycardia, a decrease in vascular tone, or both at once." Dorland's at 1818.

[5] Postural orthostatic tachycardia syndrome (POTS) is "a group of symptoms (not including hypotension) that sometimes occur when a person assumes an upright position, including tachycardia, tremulousness, lightheadedness, sweating, and hyperventilation; this is seen more often in women than in men, and the etiology is uncertain." Dorland's at 1844.

On December 6, 2012, petitioner filed a Statement of Completion.

On March 19, 2013, respondent filed his Rule 4(c) Report, recommending against compensation.

On June 11, 2013, petitioner filed a status report regarding her review of video footage and attached a list of those videos which she contended were relevant and irrelevant. Videos included interviews on the television shows "20/20," "Inside Edition," "60 Minutes," "NBC Washington," "Fox D.C. News," and "NBC Charlotte News," videos of petitioner incapable of walking forward and then running races, and multiple videos during the week she spent at Dr. Buttar's treatment center in North Carolina.

On July 7, 2013, former Special Master Zane issued a decision awarding interim attorneys' fees and costs to petitioner's former attorney, stating that the special master concluded a reasonable basis existed only up to the point of petitioner's former counsel's withdrawal. "Whether a reasonable basis existed beyond this point and continues to exist to date cannot be decided based on the record at present, and this decision should not be construed as making any such decision." Int. Fees Dec. at 5 n.7. Former Special Master Zane awarded petitioner's former counsel $44,961.65 in fees and costs. Id. at 8. Judgment entered on August 2, 2013.

On September 4, 2013, this case was reassigned to former Special Master Lisa Hamilton-Fieldman.

On October 22, 2013, petitioner filed the expert report of Dr. Lawrence Steinman, a neurologist. Ex. 65. This was over two years after she filed her petition.

On April 25, 2014, respondent filed the expert report of Dr. Peter D. Donofrio. Ex. B.

On May 30, 2014, former Special Master Hamilton-Fieldman issued a decision awarding petitioner's interim costs of $2,950.29. Judgment entered on July 1, 2014.

On August 14, 2014, petitioner filed the first supplemental expert report of Dr. Steinman. Ex. 92.

On August 19, 2014, former Special Master Hamilton-Fieldman issued an Order for petitioner to file a second supplemental report from Dr. Steinman, explaining how demyelination led to the variety of petitioner's symptoms.

On August 28, 2014, petitioner filed Dr. Steinman's second supplemental report. Ex. 108.

On January 13, 2015, respondent filed the expert report of Dr. Eric Lancaster, a neurologist. Ex. H.

On January 15, 2015, the case was reassigned to the undersigned.

On February 20, 2015, respondent filed the expert report of Dr. J. Lindsay Whitton. Ex. Z.

On June 1, 2015, petitioner filed a third supplemental report of Dr. Steinman. Ex. 118.

On August 11, 2015, respondent filed a Motion for Issuance of Subpoena to obtain any and all documentation, video files, audio files, news releases, reporting, or broadcasts, and/or website postings/updates relating to petitioner posted on Dr. Buttar's websites. On the same date, the undersigned granted respondent's motion.

On September 4, 2015, respondent filed the first supplemental expert reports of Dr. Lancaster (Ex. RR) and Dr. Whitton (Ex. SS).

On October 13, 2015, petitioner filed a supplemental declaration in support of her petition (Ex. 139). She explains how she became a media star by saying she alerted her family and friends about her physical problems. Ex. 139, at 4. One of her former colleagues was working for the county newspaper and asked if he could do an article in his paper explaining her condition and its being tied to flu vaccine. Petitioner writes she agreed since he was a friend and struggling to get started in his new job. Petitioner writes:

> After I learned that the flu vaccine was causing my issues, I thought I was doing a public service by speaking out as perhaps the batch of vaccine I received was tainted. From there the media circus began, starting with local stations contacting me to national syndicates. However, I remember one of my doctors or psychologists recommending later on that I should avoid the media as the chaos was probably not helping my symptoms, but likely contributing to them.

Id.

Petitioner continues in her supplemental declaration by saying that she thought her first attorney Robert Krakow, whom her then-husband hired per Stan Kurtz's recommendation, was involved to help her deal with media relations. Id. She continues:

> At this time, I was also contacted by Stan Kurtz from Generation Rescue, who is an anti-vaccine advocate. He and his organization quickly commandeered my injury to turn it into a poster story for their cause against vaccines[.] [I]n exchange they promised to "cure" me with the help of Dr. Buttar. Upon the advice of Dr. Buttar, I declined Dr. Cintron's plasmaparesis [sic] recommendation and started receiving [Dr. Buttar's] protocol of several bags of IV hydration mixed with his own cocktail of vitamins. This began to relieve quite a bit of the dizziness and fainting I had when I stood up or ate. Slowly, my speech started to improve and I could eat, but limited amounts of food. . . .

Id. at 4-5. Petitioner then goes through the videos respondent's neurologic expert Dr. Lancaster saw and about which he comments in his first expert report (Ex. H). Petitioner gives her own interpretation of them. Id. at 8-25. She mentions giving an interview to channel 5 and stating she could not walk or talk normally and the only thing she could do was run. Id. at 10. She

states her voice returned when she was running. She told John Henry of Channel 5 that when she runs, her heart rate would go down to 60, whereas when she walked, her heart rate was in the 130s. Id. at 10.

She mentions that Dr. Buttar talked about getting thousands of patients after airing on television petitioner's treatment with him. Id. at 23. He talked about people paying $500-600 for his drops. Petitioner states she is not sure how the drops work, but they did seem to improve her muscle weakness, but much faster than her myasthenia drugs do because of the drops' rapid absorption through her skin. Id. Petitioner describes getting an EEG with Ms. Preston, whom she refers to as "Dr." Preston even though she is a Ph.D., not a medical doctor. Id. at 24. Petitioner says Dr. Preston determined petitioner was having seizure. Id.

On January 13, 2016, petitioner filed a fourth supplemental report of Dr. Steinman. Ex. 141.

On April 26, 2016, respondent filed a CD with Exhibits AAA-JJJ, consisting of the following: Ex. AAA, "20/20" broadcast, approximately July 25, 2010; Ex. BBB, "Inside Edition Update," February 4, 2010; Ex. CCC, "20/20" broadcast followed by Robert Scott Bell show; Ex. DDD, [D.G.] update, October 29, 2009; Ex. EEE, "NBC Washington," October 15, 2009; Ex. FFF, "Fox 5 DC News," October 15, 2009; Ex. GGG, "Inside Edition," October 16, 2009; "Ex. HHH, "Fox 5 DC News," October 19, 2009; Ex. III, "NBC Charlotte," November 5, 2009; and Ex. JJJ, "Fox 5 DC News," November 19, 2009. [6]

From June 14-17, 2016, the undersigned held a four-day hearing. On the second day of the hearing, petitioner sank from her chair to the floor in the hearing room, making growling noises. The undersigned asked petitioner's expert Dr. Steinman to attend to her while the undersigned's law clerk called 911. Petitioner was transported by ambulance to MedStar Georgetown University Hospital, which necessitated the obtaining of those hospital records and the subsequent opinions of the experts interpreting those records after the hearing.

On July 14, 2016, respondent filed the second supplemental expert report of Dr. Whitton. Ex. YYY.

Also, on July 14, 2016, petitioner filed the fifth supplemental expert report of Dr. Steinman. Ex. 181.

On September 7, 2016, petitioner filed her second supplemental declaration (Ex. 190). Attached to her declaration is a copy of e-mail she sent to her treater Dr. Joey R. Gee consisting of her question and his response (Attachment 2): [D.G.] "Given the positive GAD[7] antibody

[6] Respondent also relied on the videos respondent identified as Respondent's Trial Exhibit 58-1 to Respondent's Trial Exhibit 58-66, which were specific videos derived from petitioner's Exhibit 58 (the external terabyte hard drive). Doc 143. Respondent relied on additional videos identified by dates from October 17, 2009 to October 22, 2009 including nine undated videos. Multiple videos come from Dr. Buttar's treatment center. Respondent's expert Dr. Lancaster identifies the videos upon which he relies as support for his opinion in his first expert report (Ex. H). Respondent added Respondent's Trial Exhibit 58-66 to the prior list of Respondent's Trial Exhibit 58-1 to Respondent's Trial Exhibit 58-65 at the hearing, noted in a filing dated June 21, 2016. Doc 147.

[7] GAD or glutamate decarboxylase or glutamic acid decarboxylase is "an enzyme of the lyase class that catalyzes

result, do you think this explains some of the spasms and tremors you saw in the videos online when you first took me on as a patient." Petitioner then says that when she tried to go off gabapentin[8] a year previously, a lot of her symptoms returned, necessitating her going back on gabapentin, and she wondered if the drug were masking her symptoms. Dr. Gee responded, "It just might…I did think about that. The spasms can be quite extreme. I am dealing with this same issue now with a new patient and her GAD have been fluctuating between 10 to over 100[;] she is on IVIG now."

On October 14, 2016, petitioner filed the sixth supplemental expert report of Dr. Steinman. Ex. 191.

On December 16, 2016, respondent filed the third supplemental expert report of Dr. Whitton (Ex. ZZZ) and the second supplemental expert report of Dr. Lancaster (Ex. FFFF).

On May 16, 2017, petitioner filed the seventh supplemental expert report of Dr. Steinman. Ex. 198.

On May 17, 2017, petitioner filed her third supplemental declaration (Ex. 205), arguing that respondent's neurologic expert Dr. Lancaster was wrong for assuming Dr. Gee told the treating doctors at MedStar Georgetown University Hospital that petitioner had a "non-organic (psychogenic)[9] gait disorder." Ex. 205, at 3. She asserts that in the five years she has been seeing Dr. Gee, he never "even insinuated" that she had an underlying psychogenic illness. Id.

Also, on May 17, 2017, the undersigned issued an Order to petitioner that she file by June 19, 2017 a statement from Dr. Gee indicating whether he believed petitioner had a high GAD antibody in 2009 and, if she did, whether the high GAD antibody could have caused some of petitioner's spasms and tremors that she manifested in 2009 in her videos. Moreover, the undersigned asked Dr. Gee to opine whether he thought petitioner had myasthenia gravis in 2009 and, if he did not think she did, then how would a purportedly high GAD antibody be connected to her spasms and tremors in 2009. Dr. Gee never provided petitioner with answers to the undersigned's questions. Therefore, the undersigned never learned if petitioner's assertions in her third supplemental declaration (Ex. 205) as to what Dr. Gee told her were accurate.

On June 19, 2017, petitioner filed the eighth supplemental expert report of Dr. Steinman, answering the questions the undersigned asked Dr. Gee to answer in her Order of May 17, 2017, even though the undersigned addressed those questions to Dr. Gee and not to Dr. Steinman. Ex. 206.

On September 15, 2017, petitioner filed an amended petition, which repeats in detail her prior allegations. She states flu vaccine caused her the following conditions: (1) autoimmune

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the decarboxylation of glutamate to form γ-aminobutyrate (GABA). The enzyme is a pyridoxal phosphate protein, and the reaction occurs within the mitochondria in kidney, and outside the mitochondria in brain. Deficiency of the brain enzyme may be the cause of convulsions that begin in infancy and are responsive to pyridoxine therapy." Dorland's at 790.

[8] Gabapentin is "an anticonvulsant that is a structural analogue of γ-aminobutyric acid (GABA), used as adjunctive therapy in the treatment of partial seizures; administered orally." Dorland's at 753.

[9] Psychogenic means "produced or caused by psychological factors. See also *psychosomatic*." Dorland's at 1549.

autonomic neuropathy/dysautonomia;[10] (2) myasthenia gravis[11] and in parenthesis states the following: petitioner admits myasthenia gravis is not her expert Dr. Steinman's "favorite" diagnosis; instead it is Dr. Geoffrey L. Sheean's diagnosis and Dr. Steinman puts a high value on Dr. Sheean's opinion; and (3) autoimmunity to GAD. Am. Pet. at ¶ 60.

On October 30, 2017, petitioner filed the ninth supplemental expert report of Dr. Steinman. Ex. 208. On the same date, respondent filed the fourth supplemental expert report of Dr. Whitton (Ex. MMMM) and the third supplemental expert report of Dr. Lancaster (Ex. QQQQ).

On February 7, 2018, petitioner filed her post-hearing brief.

On May 31, 2018, respondent filed his responsive post-hearing brief.

On July 2, 2018, petitioner filed her reply post-hearing brief.

Petitioner filed 209 exhibits and respondent filed 80 exhibits, for a total of 289. The undersigned has read all of them and the entire 1,110-page transcript. The undersigned has weighed the conflicting opinions of the experts and observed their demeanor at trial. The undersigned has seriously considered the opinions of the 58 treating/diagnosing doctors and two psychologists.

Because the evidence in this case as well as the more persuasive opinions of respondent's experts and supporting medical literature show that petitioner did not have an adverse reaction to flu vaccine, the undersigned **DISMISSES** this case.

FACTS

Prevaccination Records

Petitioner was born on December 23, 1983. She is 35 years old.

Prior to vaccination, petitioner was generally healthy without significant medical problems. She had a medical history of bronchitis, nose surgery, bulimia,[12] and breast augmentation surgery. Med. recs. Ex. 2, at 11-12; Ex. 9, at 2-3; Ex. 21, at 1-2; Ex. 22, at 24-26.

On November 29, 2004, petitioner saw Dr. Michael Rodriguez of Broadlands Family Practice, complaining of a fungus for the past week or two. Med. recs. Ex. 11, at 33. Petitioner

[10] Dysautonomia is "malfunction of the autonomic nervous system." Dorland's at 575.

[11] Myasthenia gravis is "an autoimmune disease of neuromuscular function due to the presence of antibodies to acetylcholine receptors at the neuromuscular junction; characteristics include muscle fatigue and exhaustion that fluctuates in severity, without sensory disturbance or atrophy. It may be restricted to one muscle group or become generalized with severe weakness and sometimes respiratory insufficiency. It may affect any muscle of the body, but especially those of the eyes, face, lips, tongue, throat, and neck." Dorland's at 1214.

[12] Bulimia is "episodic binge eating usually followed by behavior designed to negate the excessive caloric intake, most commonly purging behaviors such as self-induced vomiting or laxative abuse but sometimes other methods such as excessive exercise or fasting." Dorland's at 259.

weighed 137 pounds. She had a temperature of 100 degrees. Her blood pressure was 130/60. Id. Dr. Rodriguez diagnosed petitioner with tinea nigra[13] (ringworm). Id. at 34.

On November 16, 2006, petitioner saw Dr. Rodriguez of Broadlands Family Practice, for tinea nigra. Id. at 32.

On July 2, 2008, petitioner saw PA-C Deirdre Ellis at Broadlands Family Practice, needing a health screening for work. Med. recs. Ex. 22, at 29. Her history included rare to occasional EtOH (ethanol). Id.

On December 16, 2008, petitioner saw Dr. Huong Thai-Kemprowski, an allergist and immunologist, for an evaluation of environmental allergies. Id. at 82. Petitioner stated she had rhinorrhea, nasal congestion, and constant sniffing. She said she had had constant nasal congestion for the prior 10 years. She has sneezing and itchy eyes in the spring. Her father had allergic rhinitis. She has post-nasal drip. Id. Physical examination of petitioner's nose showed enlarged turbinate with nasal obstruction left greater than right. Id. at 83. Test results showed no sensitivity to tree pollens, grass pollens, weed pollens, dust mites, cockroach, cat, dog, horse, and mold spores. Dr. Thai-Kemprowski's impression was there was no evidence of allergic rhinitis. Id.

In March 2009, petitioner became a Washington Redskins Cheerleader Ambassador[14] as a public relations representative for the Washington Redskins. Med. recs. Ex. 31, at 2.

On April 10, 2009, petitioner and her then-husband saw psychologist Christine M. Cosgrave for petitioner's then-husband's psychotherapy. Med. recs. Ex. 187, at 1, 3.

On May 14, 2009, petitioner saw Dr. Elizabeth Mann at Broadlands Family Practice, complaining of headache, sore throat, and chills. Med. recs. Ex. 31, at 27. Dr. Mann diagnosed petitioner with allergic rhinitis. Id. at 28.

On May 18, 2009, petitioner saw Dr. Rodriguez at Broadlands Family Practice, complaining of moderate to severe cough for several days. Id. at 25. She felt tired, achy, and had trouble sleeping. Id. Dr. Rodriguez diagnosed her with acute bronchitis. Id. at 26.

On June 22, 2009, petitioner went to Dr. Rodriguez, complaining of moderate to severe cough for several days. Id. at 23. She felt tired, achy and had trouble sleeping. Id. Dr. Rodriguez diagnosed petitioner with acute bronchitis. Id. at 24.

Postvaccination Records

[13] Tinea nigra is "a minor fungal infection, caused by *Hortaea werneckii*, having dark lesions that look like spattered silver nitrate on the skin of the hands or occasionally other areas." Dorland's at 1930.

[14] "Selected during the Redskins Cheerleaders auditions process every April, the Ambassadors' main focus is interacting with fans during all Redskins home games at FedExField. While the Redskins Cheerleaders captivate the 90,000+ fans with energetic dance routines, the Ambassadors are in the AAA Ultimate Fan Zone, Touchdown Club and Suites—and even in the stands—bringing a personal, up-close interaction with fans." Washington Redskins Cheerleader Ambassadors, ULTIMATE CHEERLEADERS, https://ultimatecheerleaders.com/tag/ambassadors/ (last visited Mar. 18, 2019).

On August 23, 2009, petitioner received flu vaccine at a Safeway Pharmacy in Reston, Virginia. Med. recs. Ex. 4, at 3.

On September 12, 2009, petitioner was transported via emergency medical services ("EMS") to the emergency department ("ED") at Inova Loudoun Hospital, complaining of weakness, overall not feeling well for the last nine days, subjective fevers, body aches, weakness, and dizziness although she worked full time and ran every day to train for a 5K race. Med. recs. Ex. 5, at 6, and Ex. 51, at 112. Petitioner reported having bronchitis four to five times since February 2009 treated with antibiotics, although she still had a productive cough and fatigue. Id. The assessment was rhabdomyolysis,[15] near syncope, recurrent respiratory issues, elevated liver function test. Med. recs. Ex. 51, at 113. Petitioner had no problem getting out of bed to a chair and her speech and thoughts were clear. Id. The nursing assessment noted petitioner was hyperventilating, complaining of fever, syncope, dizziness, nausea, non-productive cough, pain in her left upper quadrant, shivering, and feeling cold. Her speech was clear and understandable. Her temperature was normal. Med. recs. Ex. 5, at 15. The onset of symptoms was sudden. She did not have any associated shortness of breath. She reported that she "was watching TV and almost passed out and began to shake." Id. at 9. She reported a history of lightheadedness, dizziness, weakness, subjective fevers, and body aches over the previous nine days. Id. at 6, 9. Petitioner said she had a sore throat for several days the prior week. Id. at 10.

On physical examination, Dr. Zachary Malachias noted petitioner was well-appearing, alert and oriented, appeared comfortable, and had a normal pulse and blood pressure, but an increased respiratory rate. Id. Neurologically, she did not have focal motor deficits, focal sensory deficits, or nystagmus.[16] She had intact cranial nerves and normal speech. She was oriented, and had normal affect, insight, and concentration. Id. Her EKG was normal. Id. Her oxygen saturation was normal. Id. at 11. Petitioner was admitted to the hospital. Id. On blood testing in the ED, petitioner's mono percentage was high at 10.9. Id. at 12. She tested negative for both influenza A antigen and influenza B antigen, indicating she did not have the flu. She tested negative for mono. Id. Her creatine kinase ("CK")[17] tested high at 12,018 U/L when the normal range is between 19-204. Id. at 13. Her myoglobin[18] tested high at 675 ng/mL when the

[15] Rhabdomyolysis is "disintegration or dissolution of muscle, associated with excretion of myoglobin in the urine." Dorland's at 1637. Myoglobin is "the oxygen-transporting pigment of muscle [which] combines with oxygen released by erythrocytes, stores it, and transports it to the mitochondria of muscle cells, where it generates energy by combustion of glucose to carbon dioxide and water." Id. at 1223.

[16] Nystagmus is "an involuntary, rapid, rhythmic movement of the eyeball, which may be horizontal, vertical, rotatory, or mixed." Dorland's at 1307.

[17] Creatine kinase (CK) is "an Mg^{2+}-activated enzyme of the transferase class that catalyzes the phosphorylation of creatine by ATP to form phosphocreatine. The reaction effectively stores the energy of ATP as phosphocreatine in muscle and brain tissue and holds the muscle concentration of ATP nearly constant during the initiation of exercise." Dorland's at 429. ATP is adenosine triphosphate. Id. at 173. Adenosine triphosphate is "a nucleotide, the 5'-triphosphate of adenosine, involved in energy metabolism and required for RNA synthesis; it occurs in all cells and is used to store energy in the form of high-energy phosphate bonds. The free energy derived from hydrolysis of ATP is used to drive metabolic reactions including the synthesis of nucleic acids and proteins, to move molecules against concentration gradients (active transport), and to produce mechanical motion (contraction of microfibrils and microtubules)." Id. at 30.

[18] Myoglobin (Mb) is "the oxygen-transporting pigment of muscle, a type of hemoprotein resembling a single

normal range is 0-62. Id. at 14. Her CKMB[19] mass tested high at 7.49 ng/mL when the normal range is 0.00-3.38. Id.

As an inpatient, petitioner reported during a History and Physical that she had bronchitis four to five times since February 2009 and was treated with antibiotics, although she still had a productive cough and fatigue. Id. at 6. Despite her symptoms, petitioner was working full-time and running every morning to train for a 5K race. Id. Petitioner had a history of eating disorder years ago. Id. Her history also includes turbinate[20] reduction, rhinoplasty,[21] and breast augmentation. She reported a questionable blood transfusion during her breast augmentation surgery. Id. Her white count was 10.9. Id. She had no acute findings on chest x-ray. Id. She was negative for flu. Id. at 7.

On physical examination, petitioner was alert and oriented. Id. She was not in acute distress. The diagnosis was rhabdomyolysis, leukocytosis,[22] and near syncope. Petitioner did not have any gross neurologic deficits. She was out of bed to a chair without difficulty. Her speech and thoughts were clear. Petitioner had recurrent respiratory issues of an ongoing cough and fatigue. Id. She had an increased respiratory rate, but she was otherwise afebrile with no abnormalities. Id. at 10. Petitioner was diagnosed with rhabdomyolysis, leukocytosis, near syncope, recurrent respiratory issues, elevated liver function tests, and a heart murmur. Id. at 7. She was admitted to the hospital for hydration with IV fluids. Id. at 7. The results of her chest x-ray on September 12, 2009 and brain CT scan were normal. Id. at 25, 26. Her chest x-ray on September 13, 2009 showed small non-specific hypodensities in her thyroid. Id. at 28. Her EKG on September 12, 2009 was normal. Id. at 31.

Petitioner was discharged on September 14, 2009. Med. recs. Ex. 5, at 2. Dr. Brian A. Hazen wrote the discharge summary stating serial laboratory tests included a CPK which was 12,018 on September 12, 2009, but 6,546 on September 13, 2009; an AST[23] which was 366 on September 12th, but 167 on September 14th; and an ALT[24] which was 102 on September 12th, but 80 on September 14th. Id. Dr. Hazen stated these test results were probably reflective of muscle damage and not a hepatic process. Petitioner's long-term goal was long-distance running. Dr. Hazen told her to get one more test of her CPK and liver function before ensuring she had completely resolved from the muscle damage of rhabdomyolysis. In the meantime, he said it

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subunit of hemoglobin, composed of one globin polypeptide chain and one heme group (containing one iron atom); it combines with oxygen released by erythrocytes, stores it, and transports it to the mitochondria of muscle cells, where it generates energy by combustion of glucose to carbon dioxide and water." Dorland's at 1223.

[19] CKMB is "CK_2 (MB) … primarily in cardiac muscle." Dorland's at 429.

[20] Turbinate is "any of the nasal conchae." Dorland's at 1991.

[21] Rhinoplasty is "a plastic surgical operation on the nose, either reconstructive, restorative, or cosmetic." Dorland's at 1640.

[22] Leukocytosis is "a transient increase in the number of leukocytes in the blood; seen normally with strenuous exercise and pathologically accompanying hemorrhage, fever, infection, or inflammation." Dorland's at 1028.

[23] AST is aspartate transaminase. Dorland's at 167. "The serum level of aspartate transaminase (SGOT) and that of other transaminases are frequently elevated in a variety of disorders causing tissue damage."

[24] ALT is alanine transaminase. Dorland's at 54. Alanine transaminase is "an enzyme found in serum and body tissues, especially in the liver. Serum enzyme activity (SGPT) is greatly increased in liver disease and also elevated in infectious mononucleosis." Id. at 43.

was all right for her to do very light, brief aerobic exercise, to stay hydrated, not to do anaerobic exercise, and to avoid excessive heat. Id. Petitioner's diagnosis was mild rhabdomyolysis. Id.

On September 17, 2009, petitioner returned to the ED at Inova Loudoun Hospital, complaining of sudden lightheadedness, blackout, and shortness of breath, noting immediate restoration of normal mental status. Med. recs. Ex. 51, at 13. Dr. Pranav Vermani did a physical examination, noting weakness bilaterally in the upper extremities and profound weakness bilaterally in the lower extremities. Id. at 14. On the same day, during a consultation with Dr. Sarbjot S. Dulai, a neurologist, petitioner reported that she had subjective fevers, chills, body aches, generalized fatigue, weakness, and intermittent lightheadedness. Id. at 21. She had shortness of breath and tingling in her feet and hands when she was hyperventilating. Id. Petitioner continued to train for a 5K race. Although she was admitted to Inova Loudoun Hospital on September 12, 2009 and discharged on September 14, 2009, she continued to have subjective chills and fevers, generalized body aches, and intermittent lightheadedness. She claimed multiple brief episodes of passing out which occurred only when she was sitting or standing. She thought her shortness of breath was related to hyperventilation, when she would get some tingling in her feet and hands. Petitioner received intravenous hydration in the emergency room. She reported some headaches without visual changes or problems with speech, swallowing, or balance. Id. Dr. Dulai noted petitioner had very minimal weakness proximally in the lower extremities. Id. at 4, 23. Petitioner's neurologic examination was otherwise unremarkable. Id. at 23. Dr. Dulai's impression was that petitioner's symptoms were due to a continuation of her viral syndrome and possibly a component of dehydration. Id.

On September 17, 2009, petitioner's AST was high at 60 U/L when the normal range is 5-40. Med. recs. Ex. 6, at 6. Her ALT was also high on September 17, 2009 at 61 U/L when the normal range is 7-56. Id. On September 17, 2009, petitioner's ANA was 1:80 when normal is less than 1:40. Med. recs. Ex. 22, at 76. On September 18, 2009, petitioner's AST returned to normal at 38 and her ALT returned to normal at 48. Med. rec. Ex. 6, at 6. On September 17, 2009, petitioner's CK was elevated at 485 U/L when the normal range is 19-204. Id. at 7. It was still elevated, but less so, on September 18, 2009 when the CK measured 236 U/L. Id. On September 17, 2009, petitioner's C-reactive protein was negative. Id. at 17.

On September 17, 2009, petitioner had a brain MRI with and without contrast. Id. at 37, and Ex. 51, at 48. Dr. Ho-Song Lee wrote the brain MRI was normal. There was no mass, hemorrhage, or extra-axial fluid collection. The gray matter, intracranial vessels and postcontrast exam were normal. Petitioner had mild left maxillary sinus membrane thickening. Id.

On September 18, 2009, petitioner had a consultation with Dr. Jeffrey S. Luy, a cardiologist, who observed normal muscle strength and tone on physical exam. She also had normal and appropriate affect. Med. recs. Ex. 51, at 25; Ex. 6, at 1-4; Ex. 22, at 58-59. Dr. Luy's impression was that petitioner's syncope was "probably vasovagal due to some relative element of dehydration," prior rhabdomyolysis which had improved, and benign systolic heart murmur. Med. recs. Ex. 51, at 25. Dr. Luy recommended discharge with fluid, Tylenol, and rest. Id. On the same day, petitioner was discharged with a diagnosis of "syncope, likely

vasovagal, history of recent rhabdomyolysis, underlying etiology not clear, possible viral illness with complaint of still being tired and fatigued" and instructions to follow up with her primary care physician ("PCP"). Id. at 4-5 and med. recs. Ex. 22, at 48.

On September 21, 2009, petitioner visited her primary care physician ("PCP") Dr. Michael Rodriguez at Broadlands Family Practice with concerns of multiple episodes of near syncope over the past couple of weeks. Med. recs. Ex. 22, at 19. She said that she would feel dizzy, nauseated, and as if she were going to pass out. Id. She asked Dr. Rodriguez to review her ED records and tests because she was convinced that her symptoms were due to Lyme disease even though a Lyme test was negative. Id. On physical examination, Dr. Rodriguez observed normal joints and muscles. Id. at 20. Dr. Rodriguez referred petitioner to an infectious diseases specialist, Dr. Sarfraz A. Choudhary, and a rheumatologist, Dr. Alexia Gospodinoff. Id.

On September 22, 2009, petitioner visited Dr. Choudhary with complaints of headache, neck pain, multiple joint pains, and pseudoseizures. Id. at 56. On physical examination, Dr. Choudhary found nothing remarkable. Id. Dr. Choudhary stated the possibilities included "psychological as [petitioner] had a history of bulimia, tick borne illness like Lyme disease, and viral illnesses like West Nile." Id. at 57. He ordered a spinal tap with Inova Loudoun Hospital to rule out any neurological etiology. Id. Dr. Choudhary also recommended counseling and reassurance, discussed different infection control precautions, and had petitioner follow up with her neurologist and cardiologist. Id. A lumbar puncture was performed on September 24, 2009 and the evaluation of lupus was negative. Med. recs. Ex. 7 at 1 and Ex. 44, at 16, 29. Petitioner's lumbar puncture showed a normal cerebrospinal fluid ("CSF") of 24 mg/dL when the normal range is 15-60. Med. recs. Ex. 7, at 2.

On September 26, 2009, petitioner went to Inova Fairfax Hospital ED, complaining of three days of unsteady gait, difficulty with speech, syncope when standing up, shakiness, constant fatigue, an achy neck, headache, and difficulty sleeping. Med. recs. Ex. 44, at 136. Petitioner told Dr. Scott Weir that she was recently discharged from Loudon Hospital on September 21, 2009 for the same symptoms and that she received flu vaccine three days before her symptoms began. Id. Petitioner arrived via stretcher since she was unable to ambulate. Id. at 137. Petitioner's speech was clear and understandable. Her bilateral hand grasp was weak and toes were progressing upward. Petitioner reported "it feels like numb spots in my mind." Id. Petitioner's family reported petitioner seemed confused at times and sometimes could not finish her sentences. Id. Her C-reactive protein was normal. Id. at 142. Petitioner said the onset of symptoms was gradual and occurred on September 3, 2009, starting with a fever and headache. She developed syncopal episodes after meals, difficulty speaking and concentrating, tremors in her neck, head, and arms, weakness in her legs, and a wide-based and unsteady gait. She said she received flu vaccine on August 30, 2009 (the wrong date; she received flu vaccine on August 23, 2009). She was admitted to Loudoun Hospital for a cardiac syncopal workup and discharged on September 21, 2009 with no definitive diagnosis. Petitioner stated she developed intermittent paresthesia[25] in her legs and arms. She developed seizure-like episodes two days previously with

[25] Paresthesia is "an abnormal touch sensation, such as burning, prickling, or formication, often in the absence of an external stimulus." Dorland's at 1383.

jerking of her arms and legs, but no postictal period or incontinence. Petitioner states she knew what was happening but could not stop her muscles. She denied slurred speech and stated she has difficulty concentrating and difficulty getting words out. Id. Petitioner reported chills, fatigue, weakness, vision changes, seeing visual spots, syncope, nausea, arthralgias, myalgias, and joint stiffness. Id.

Physical examination showed petitioner was ill-appearing and uncomfortable. Id. at 143. The range of movement of her neck was within normal but difficult due to tremulous movements and stiff motions. She had a tremulous head. Her extremities had 4/5 strength. She had dysmetria[26] on cerebellar examination. Petitioner was admitted to the hospital for neurologic examination. Id.

On admission, petitioner complained of a four-day history of worsening and bilateral lower and upper extremity weakness, difficulty ambulating, and generalized tremors with multiple vague somatic complaints. Med. recs. Ex. 9, at 1-2; Ex. 44, at 1 (complaining of syncope, low blood pressure, with an admitting diagnosis of other malaise and fatigue); Ex. 22, at 50. She complained of having "sweating" in the central core area and mid-epigastric pain that stopped beneath the sternum. Med. recs. Ex. 44, at 55. She was discharged on September 29, 2009 with a principal diagnosis of abnormal involuntary movement not elsewhere classified ("NEC"), and secondary diagnoses of lack of coordination, other malaise and fatigue, conversion disorder,[27] obstructive sleep apnea, and stuttering. Med. recs. Ex. 44, at 8.

Dr. Mohammed A. Mannan was petitioner's attending physician. Med. recs. Ex. 9, at 1. Petitioner told Dr. Mannan that she had been in excellent health until around August 30, 2009 (which would be one week after she received flu vaccine). Id. She said within a few days after getting a flu vaccination, she had a flu-like illness with upper respiratory symptoms, bronchorrhea,[28] sore throat, mild nonproductive cough, fevers, chills, and diffuse myalgias. She had some lightheadedness, dizziness, and syncopal episodes and presented to Inova Loudon Hospital. (She went to Inova Loudon Hospital on September 12, 2009.) Id. Dr. Mannan notes in petitioner's records that petitioner had a positive ANA "which was only 1:80." Id.

[26] Dysmetria is "a condition in which there is improper estimation of distance in muscular acts, with disturbance of the power to control the range of muscular movement, often resulting in overreaching." Dorland's at 578.
[27] Conversion disorder is "a mental disorder characterized by conversion symptoms (loss or alteration of voluntary motor or sensory functioning suggesting physical illness, such as seizures, paralysis, dyskinesia, anesthesia, blindness, or aphonia) having no demonstrable physiological basis and whose psychological basis is suggested by (1) exacerbation of symptoms at times of psychological stress, (2) relief from tension or inner conflicts (primary gains) provided by the symptoms, or (3) secondary gains (support, attention, avoidance of unpleasant responsibilities) provided by the symptoms. Many patients exhibit "la belle indifference," a lack of concern about the impairment caused by the symptoms; histrionic personality traits are also common. Symptoms are neither intentionally produced nor feigned, and are not limited to pain or sexual dysfunction." Dorland's at 549. Dyskinesia is "distortion or impairment of voluntary movement, as in tic, spasm, or myoclonus." Id. at 578. Myoclonus is "shocklike contractions of a portion of a muscle, an entire muscle, or a group of muscles, restricted to one area of the body or appearing synchronously or asynchronously in several areas." Id. at 1222. Aphonia is loss of voice, mutism. Id. at 115.
[28] Bronchorrhea is "excessive discharge of mucus from the bronchi." Dorland's at 253.

Petitioner complained that she had progressive deterioration in the prior four days and some new symptoms, including symmetric lower greater than upper extremity weakness and difficulty ambulating, as if her legs were going to buckle due to weakness. She was much more fatigued and very easily winded with minimal exertion. However, she denied any shortness of breath at rest, but stated she got very exhausted and short of breath with any minimal exertion. She reported difficulty sleeping. She stated she had onset of worsening uncontrollable tremors at times that became worse with effort or exertion. She had intermittent headaches, but no vision changes. She stated that, at times, she had trouble concentrating and had stuttering speech, but not specific slurring. She denied the following: difficulty swallowing, chest pain, recent cough, palpitations, nausea, vomiting, abdominal pain, diarrhea, constipation, urinary changes, calf pain, swelling, orthopnea, or PND (paroxysmal nocturnal dyspnea[29]). She described occasional shooting tingling pains in her lower extremities more than in her upper extremities which was symmetric. She stated her sensation was intact. She reported intermittent, mainly frontal but occasionally diffuse, headaches, giving a vague description of occasionally feeling cold spots in the back of her head. She reported some fainting episodes and occasional lightheadedness which was somewhat worse when she was upright. She described recent fevers as occasional hot flashes but did not measure them. She said she received a flu vaccine two to three years ago without problems. Id. at 2. Her past medical history included chronic bronchitis, bulimia, obstructive sleep apnea with turbinate reduction around 2008, rhinoplasty, breast augmentation, intestinal surgery in early childhood, and oral and genital herpes. Id. at 2-3. A chest x-ray done on September 26, 2009 because petitioner complained of shortness of breath was stable. Dr. Elise Berman noted petitioner had degenerative change to her right AC (acromioclavicular) joint. Id. at 7.

On physical examination, petitioner's blood pressure was 110/60 and her pulse 70. Id. at 3. She was alert and oriented, well-nourished, somewhat anxious, and generally fatigued, but generally comfortable. She was very easily fatigued with minimal activity. Id. She did not have a heart murmur. Id. at 4. Her C3 and C4 complement levels were normal at 85 and 15 respectively.

Dr. Mannan's impression was multiple somatic complaints following flu vaccination initially with flu-like illness with myalgia, rhabdomyolysis, and mild transaminitis (high level of enzymes), all of which resolved. She had recurrent near syncope and syncope. Id. Petitioner did not lose her distal deep tendon reflexes. Conversely, she complained of generalized tremors and possible hyperreflexia, more so in the upper extremities and worse with effort. Dr. Mannan wrote that "Etiology regarding above constellation of symptoms is not entirely clear at present." Id.

On September 27, 2009, Dr. Jonathan Bresner, a neurologist, saw petitioner because of abnormal movements. Med. recs. Ex. 44, at 29. Petitioner said that on September 23, 2009, she had shooting pains in various parts of her body and sensory changes in her head. She felt as though her whole body was hot although her hands and feet were cold. She described seeing white spots in her vision and had intermittent headaches. She had lab work done and the only

[29] Dyspnea is "breathlessness or shortness of breath; difficult or labored respiration." Dorland's at 582.

abnormality was an ANA of 1:80. Petitioner's lumbar puncture was normal. Id. A family friend who is a physician was concerned petitioner had GBS because of her difficulty breathing, which is why she returned to the ED. Id. at 29-30. She had a history of bronchitis five times in the last year and going to the ED for severe abdominal pain. Id. at 30. On physical examination, petitioner had fluent speech, but also stuttering and halting speech. She was alert, oriented, and anxious. Her strength was 5/5 throughout. Her tone was alternating and uncoordinated with tremors, contractions, dystonic[30] posturing, and myoclonic jerks. She had spastic jerking movements of the limbs and at times dystonic posturing as well. Her reflexes were 1 to 2+. She was able to walk unassisted, but had jerking movements of her entire body while she was walking. Id.

Petitioner's lab results were normal for CK, C-reactive protein, and erythrocyte sedimentation rate. Id. at 31. The protein in her CSF was 24. Dr. Bresner's impression was multiple complaints progressing over the last month as well as progressive tremulousness and abnormal body movements for the prior several days. Dr. Bresner states:

> The observed movements are extremely peculiar and not easily described from a neurological standpoint. It is also odd that the patient's symptoms resolved for a long enough period each morning for her to apply makeup but then return so forcefully that she is unable to speak or move her limbs in any sort of coordinated motion. The patient is able to walk despite her inability to control her limbs . . . . I am reassured that her neurological exam is otherwise normal and had an extensive workup including laboratory CSF and reportedly a brain MRI are also unremarkable, excluding a borderline elevated ANA. … I was originally asked to assess the patient for Guillain-Barre syndrome. There is [sic] currently no signs or symptoms on her exam to suggest this diagnosis. A psychogenic etiology to the patient's symptoms remains a consideration. The patient might benefit from psychological counseling or psychiatric evaluation. . . .

Id. at 31-32.

On September 28, 2009, petitioner had a psychiatric consultation on a question of conversion disorder with Dr. Paul M. Dellemonache. Med. recs. Ex. 21, at 1-6, and Ex. 44, at 33-38. Petitioner reported that she had a history of bulimic and self-induced vomiting behavior during her teens "to control something since my father controlled me." Med. recs. Ex. 21, at 2. Neurology had seen petitioner and did not feel her signs and symptoms were consistent with a clear neurologic etiology and questioned a psychogenic element. Id. at 1. The only stressor petitioner could mention within the past year was her younger sister's suicide attempt. Id. Petitioner felt her sister did that to get attention as she had apparently done before. Petitioner stopped speaking with her sister who had since moved out of petitioner's house and back in with

[30] Dystonia is "dyskinetic movements due to disordered tonicity of muscle." Dorland's at 582.

petitioner's father. Petitioner reported her other siblings agree with her regarding her sister, but denied that this caused any difficult family strife. Petitioner did not speak to her mother after her parents' separation when she was 18 years old. Id. Petitioner thought her mother was jealous of petitioner's relationship with her father. Id. at 3. She was not distressed when her parents separated because she felt her father could do better. Petitioner moved out of the house when she was 18 to her own house which she then flipped and moved into a bigger house but lost its equity when the housing market declined. She went straight to work at AOL after high school. In order to get onto the Washington Redskins cheerleading team, she had breast augmentation which she felt was a necessity to get onto the team. She practices once or twice a month and enjoys it. She also enjoys marathon running. Id. Petitioner noted that her illness had brought her then-husband and her "even closer together." Id. at 5.

Petitioner said she had bronchitis several times over the prior year and thought her symptoms might be due to Lyme disease because she had several coworkers and friends who themselves or their family members had Lyme disease with similar presentations and she had many tick bites on her legs in the past from running outside. Id. at 2. She also mentioned that she had a flu vaccination at the end of August which might or might not be related to her symptoms. Id. Dr. Dellemonache noted that conversion disorder is a "diagnosis of exclusion," which could not be conclusively diagnosed until all other workups were exhaustive and continued to be negative. Id. A lupus anticoagulant evaluation done on September 28, 2009 resulted in no detection. Med. recs. Ex. 8, at 6, 9. Testing for Epstein-Barr virus antibody and IgG was positive, but IgM was negative. Id. at 10. Cytomegalovirus was not detected. Id. at 16.

On September 28, 2009, petitioner and her then-husband met with a social worker Michelle Ougheltree. Med. recs. Ex. 44, at 57. Petitioner still had a significant stutter. Petitioner was assisted in walking that morning by two persons because her knees buckled and currently her knees and hips hurt. Petitioner was interested in outpatient therapy. Petitioner's then-husband requested SW Ougheltree complete a short-term disability form as petitioner's sick leave had run out. The social worker would give the form to petitioner's PCP. Id.

On September 29, 2009, petitioner was discharged. Dr. Mannan diagnosed her with "multiple somatic complaints and progressive but fluctuating neurologic deficits (including abnormal movements/tremors and speech) of unclear etiology, not fitting any particular pattern. Possible psychogenic etiology. No evidence of GBS." Id. at 13. He also said that petitioner's orthostatic hypotension was likely due to volume depletion and it improved with intravenous hydration. Dr. Mannan writes that at the time of petitioner's presentation, she had "very odd neurological symptoms with difficulty walking and tremors during evaluation of strength." Id. However, petitioner's reflexes were normal, and she did not have any focal findings. Id. Dr. Bresner in the ED suspected petitioner's neurological symptoms were not consistent with GBS and there could be a psychogenic component to her symptoms. Id. at 13-14.

In his discharge summary, Dr. Mannan drew attention to the fact that petitioner "was unable to hold a cup of water in her hand without spilling; however, she was able to fully put makeup on in the morning during hospitalization, including eyeliner, without complication until confronted by the nursing staff, after which she no long could do this task." Id. at 14. All of

petitioner's lab tests, including Lyme disease, repeat ANA, antiphospholipid antibody panel, West Nile, and H_1 N_1, were negative. Id. Dr. Mannan discussed with petitioner's then-husband putting petitioner on doxycycline even though multiple tests for Lyme disease were negative. Her then-husband said petitioner had been very concerned about possible Lyme disease after researching it on the internet and he felt petitioner would be greatly reassured by empiric treatment for it. Dr. Mannan gave petitioner a one-week course of doxycycline upon discharge. In addition, petitioner was treated with IV hydration to help with orthostatic and volume depletion during this hospitalization. Id. Dr. Mannan spoke with petitioner's PCP Dr. Rodriguez who also did not see any clear evidence of obvious organic etiology, and seemed concerned about a psychogenic etiology, and agreed with a psychiatric evaluation. Id. Petitioner requested a note for short-term disability but Dr. Mannan suggested she follow up with Dr. Rodriguez because there was no clear diagnosis. Id. Lab results showed a negative C-reactive protein and a sedimentation rate of 19. Her cortisol at 10.7 was normal as was her B-12. ANA was detected with a ratio of 1:80. Lyme antibodies were negative. Med. recs. Ex. 22, at 49.

On October 2, 2009, petitioner saw Dr. Garry Ho at Broadlands Family Practice, for a consultation and to review her recent medical problems. Med. recs. Ex. 22, at 15-17. She discussed multiple sclerosis ("MS") with Dr. Ho. Id. at 15. Petitioner reported that she woke up on September 24, 2009 with trouble walking "like I had MS." Id. She also reported that she developed dysarthria and stuttering when she tried to talk out loud on September 26, 2009. Id. At that time, petitioner was in the process of setting up an admission to Johns Hopkins Hospital's neuromuscular neurology service. Id. On physical examination, Dr. Ho found petitioner had a broad-based, spastic, stamping and waddling gait, and dysarthric and stuttering speech. Id. at 16-17.

On October 2, 2009, petitioner went to Johns Hopkins Medicine ED and spoke to Dr. Julius C. Pham. Med. recs. Ex. 55, at 24. Johns Hopkins was to evaluate whether petitioner had GBS since she reported lower leg weakness that progressed to her upper legs, increased difficulty speaking and walking, and shortness of breath. Id. She had uncontrollable bobbing of her head when speaking or making intentional movements. She also described electric-like shooting pains starting in her legs and moving around her body. She also had symptoms of dysphonia that whispering or singing in a high-pitched voice relieved. She had difficulty using her lower limbs for prolonged periods of time because either they gave out or she had uncontrollable movements in them. Id. Petitioner complained of a headache at the base of her neck which was constant and dull. Id. When it went away, she had diffuse, cool spots all around or she developed a headache behind her right eye and again had cool sensations. Id. at 24-25. Petitioner said she had episodes of uncontrollable blinking that she was unaware of and photophobia. Id. at 25. She had mild congestion and intermittent tinnitus in her left ear that woke her. She had dysgeusia.[31] Petitioner had inappropriate episodes of uncontrollable laughter. She had autonomic dysfunction with hot flushing at the core and cool extremities. She had syncopal episodes associated with eating, and loss of consciousness without incontinence or tongue biting after she had bobbing of her head. Her sleep cycle was off. She had increased

[31] Dysgeusia is parageusia. Parageusia is "a bad taste in the mouth." Dorland's at 577, 1375.

appetite and a recent two-pound weight loss. Warm water worsened her symptoms, particularly at her knees. Petitioner told Dr. Pham she received flu vaccine on August 30, 2009 (not the true date of August 23, 2009). She was taking doxycycline and prednisone.[32] Id.

On physical examination, petitioner did not have nystagmus. Id. Her lower extremities were cool to the touch. Id. at 26. On motor examination, petitioner had an abnormal, ataxic, waddling/wide stance. Her strength was 4/5 in the upper and lower extremities. Her right upper extremities were weaker, but her grip was equal. Petitioner tapped her foot and her head bobbed. Dr. Pham's final assessment was that petitioner presented with multiple neurologic findings both central and peripheral in nature. The final diagnosis was weakness. Dr. Pham wrote petitioner had a complex problem, which was likely neurological, but for which she had not received a diagnosis even though several outside medical facilities and neurologists had evaluated her. About one month ago, she was normal. Since then, she developed lower extremity weakness, paresthesia, and some dysphonia. The differential diagnosis included GBS, MS, Lyme, and myasthenia gravis. Id. Petitioner awaited admission to neurology for unexplained ascending weakness and head bobbing. Id.

A nursing assessment dated October 2, 2009 notes that petitioner was brought to a room by wheelchair with ED staff. Id. at 27. Petitioner told RN Michelle E. Charron that she had pain in both ears when she tried to speak. Petitioner said she had difficulty breathing "like I can't get enough oxygen." Id. She said she had nausea after eating and weight loss despite increased food intake. She had 5/5 muscle strength throughout. She had severely stuttering speech which began one week previously. She reported progressive weakness in her lower extremities moving up her legs. Id. RN Pia L. Bolano did a reassessment, during which petitioner denied shortness of breath, and denied nausea, vomiting, diarrhea or abdominal pain. Id. RN Czereyna C. Pearl similarly wrote petitioner denied shortness of breath, nausea, vomiting, diarrhea or abdominal pain. Id. at 28. RN Pearl noted petitioner's positive stuttering. Id. at 28. A chest x-ray done on October 2, 2009 was normal. Id. at 33. On October 2, 2009, petitioner had an MRI of her lumbar spine which did not show abnormal enhancement within the cervical, thoracic, or lumbar spinal cord or cauda equina. Id. at 18. She had mild degenerative changes including a T7-T8 disc bulge and annular tears in the L4-L5 and L5-S1 discs. Id.

On October 3, 2009, petitioner was admitted to Johns Hopkins Hospital. Med. recs. Ex. 2, at 11. Dr. Anjajl Sharrief took a history that petitioner received flu vaccine on August 30, 2009 (not the true date of August 23, 2009). Three days afterward, she woke with a sore throat and congestion, progressing to fever and fatigue. She then had severe fatigue and muscle aches but continued to work. She came to the emergency department on September 12, 2009 with generalized weakness and lightheadedness. On the day she presented, she reportedly had generalized convulsions and an episode of syncope. Her CK was 12,000 on presentation with MB of 7.5. Myoglobin was 75. Troponin was negative. AST and ALT were elevated at 366 and 102 respectively. Her white blood cell count was 10.6 on admission. This was thought to be secondary to a viral illness. CK and liver function tests came down with IV fluid hydration. She

[32] Prednisone is "a synthetic glucocorticoid derived from cortisone, administered orally as an anti-inflammatory and immunosuppressant in a wide variety of disorders." Dorland's at 1509.

was admitted from September 12 to 14, 2009 and discharged home. She went back to work on September 17, 2009 and began feeling weak with nausea and fainting at work. She began trembling uncontrollably and was readmitted to the hospital. She was discharged the following day.

Since her lethargy continued, she saw her PCP on September 21, 2009 who said petitioner's ANA was positive at 1:80 and that she had systemic lupus erythematosus ("SLE").[33] Petitioner began having reproducible chest pain and was referred to an infectious disease specialist and a rheumatologist. When she saw the infectious disease specialist on September 22, 2019, she fainted with convulsions. The doctor had a lumbar puncture performed. Petitioner began having difficulty ambulating with knees buckling and continued nausea, chills, sweats, and lightheadedness. She described that at this point she began having vivid dreams and difficulty sleeping. Id. Petitioner also began having headaches which she told Dr. Sharrief felt like cold spots in the back of her head. She returned to the hospital on September 27, 2009 with unsteadiness and difficulty speaking. She described it as pain in her face and neck when she spoke. She sounded as if she were stuttering. She could talk normally if she whispered. She began to have symptoms in her toes which petitioner described as their moving erratically and misfiring. Several specialists whom she saw could not determine the etiology of her symptoms. She began taking doxycycline for possibly Lyme disease. She also started taking methylprednisolone[34] Dosepak for chest pain which her PCP prescribed. Petitioner's symptoms persisted and progressed to intermittent uncontrollable blinking, difficulty focusing, pain in her neck muscles, uncontrollable shaking, cold feelings in her feet, and sharp pain in her legs. She was referred to Johns Hopkins ED. Id.

Petitioner's history was rhinoplasty, breast augmentation, and oral and genital herpes. Id. at 12. She has a half-sister who had an autoimmune disorder at age four. Her cousin has Grave's disease and now breast cancer. Her maternal great aunt has multiple sclerosis. Her mother's father's mother had fibromyalgia. Her maternal grandmother had breast cancer. Her maternal grandfather had lung cancer. Her father has a benign brain tumor. Petitioner said she has possible tick bites from a 5K run in August 2009. Id.

On physical examination, Dr. Sharrief noted that petitioner was well-appearing, sitting up in a stretcher in no apparent distress. Her face was "very well made up." Id. Her heart rate was 77, and blood pressure 111/63. Neurologically, petitioner was spontaneously alert and oriented. She was able to give a full history which was limited only by her inability to speak properly. She had good attention and a normal fund of knowledge. She registered 4 out of 4 items and recalled 4 out of 4 after five minutes. Id. Petitioner spoke in a broken voice taking deep gasps of air between words. Id. at 13. Her speaking appeared labored. She was able to whisper without a

[33] Systemic lupus erythematosus is "a chronic, inflammatory, often febrile multisystemic disorder of connective tissue that proceeds through remissions and relapses; it may be either acute or insidious in onset and is characterized principally by involvement of the skin …, joints, kidneys, and serosal membranes." Dorland's at 1080.
[34] Methylprednisolone is "a synthetic glucocorticoid derived from progesterone, used in replacement therapy for adrenocortical insufficiency and as an anti-inflammatory and immunosuppressant in a wide variety of disorders." Dorland's at 1154.

broken voice. Petitioner explained this by saying speaking in a normal voice caused a strain on her face and neck muscles.

Petitioner's shoulder shrug was 5 out of 5. She had normal bulk and tone. She had a jerky vertical tremor of her head which waxed and waned in intensity depending on her level of exertion. Tremor also affected her arms and legs. Her strength was 5 out of 5 proximally in the upper extremities including deltoids, biceps, and triceps. Petitioner had mild weakness (4+ out of 5) in wrist extension, finger extension, and finger flexors on the left hand. She was strong in these groups on the right. Her lower extremity strength was notable for some component of giveaway weakness. Id. Her reflexes were 2+/brisk in her bilateral biceps, triceps, brachioradialis, patellae, and Achilles. She did not have clonus. Her toes were downgoing bilaterally. She had intact and symmetric sensation to light touch and temperature proximally and distally. Her vibratory sensation was intact. Her finger-nose-finger coordination was slow and brought out severe vertical head tremor as did heel-to-shin testing. She was able to stand without assistance. Her gait was narrow-based. After taking 2-3 steps, she began to have severe shaking of her head in a vertical motion and then a bouncing motion of her legs as if her knees were going to give way. She asked to sit down. Id.

Petitioner's lab studies showed erythrocyte sedimentation rate on September 26, 2009 of 12, negative results for: ANA, anti-double stranded DNA, lupus, Epstein Barr virus ("EBV") IgM. Her EBV IgG was greater than 5, and her EBV viral capsule antigen IgG was 3.8 high. On September 17, 2009, her C-reactive protein was low titer negative, cortisol was 10.7, ANA was detected at 1:80, and HIV western blot negative. A lumbar puncture on September 24, 2009 showed zero white blood cells, and a protein of 24. Her Lyme antibodies were negative. Id. On October 4, 2009, petitioner's erythrocyte sedimentation rate was normal at 6 mm/hr when normal is 4-25. Med. recs. Ex. 55, at 66. On October 4, 2009, petitioner's ANA was positive at 1:320. Her Lyme disease antibody was negative. Id.

Petitioner's September 17, 2009 brain MRI was normal with no mass hemorrhage or extra-axial fluid. She had mild left maxillary sinus membrane thickening. A chest CT on September 13, 2009 showed soft tissue in the anterior mediastinum probably residual findings without mass effect. A head CT done on September 12, 2009 was normal. Dr. Sharrief reviewed the September 17, 2009 brain MRI. The T1 and T2 images showed no signal abnormality. Id. There was no evidence of masses, ischemic stroke, bleeding, or a demyelinating process. Med. recs. Ex. 2, at 14.

Dr. Sharrief's assessment was petitioner did not have significant medical problems until four weeks before she came to Johns Hopkins. Her presentation began with what seemed like a viral illness. She had constitutional symptoms and upper respiratory symptoms. She was admitted to another hospital and found to have elevated CK and elevated liver function tests. These tests eventually normalized. She had progressive symptoms including some lightheadedness, many episodes of syncope, tremor in her head, pain in her neck and face which affected her voice, and inability to ambulate because of perceived weakness in her legs. An extensive workup included brain MRI, lumbar puncture, and multiple laboratory studies, none of which were revealing. Her ANA was positive with a low titer of 1:80. Lyme studies were

negative. Inflammatory markers were not elevated. She began on doxycycline and prednisone, but there was no evidence to begin these drugs. Petitioner's neurologic examination had many components which were not physiologic. Nevertheless, it was possible that her viral syndrome had persistent effects which debilitated her. She could have had exposure to ticks within weeks of her initial presentation. Tickborne illness might explain some of her symptoms, but not many of her neurologic complains. Dr. Sharrief thought there was currently a likely component of "psychological overlay contributing to her symptoms." Id.

Dr. Victor C. Urrutia, a neurologist, assessed that petitioner's symptoms clearly had a strong psychogenic component but noted that petitioner did have elevated liver enzymes and CPK, and could have had a mild post-vaccination reaction with neurological symptoms. Id. He reassured petitioner that she did not have GBS and there was no evidence that she had SLE or Lyme. He recommended she stop taking doxycycline and prednisone. He thought if the workup were negative, she might have had a mild reaction and she would get better soon without specific treatment. Petitioner told him she saw a psychologist that ruled out stress. Petitioner was admitted because she could not walk. Id. and med. recs. Ex. 55, at 12. A neurology progress note dated October 3, 2009 states astasia-abasia.[35] Med. recs. Ex. 55, at 85.

On October 3, 2009, petitioner had a brain MRI, which did not show an acute process or abnormal enhancement. Id. at 15.

On October 4, 2009, Dr. Urrutia further evaluated petitioner. Med. recs. Ex. 2, at 10. Petitioner stated she felt better and was able to walk backwards and sideways but still not forward. A physical therapist mentioned dystonia to her. After receiving Ativan[36] the prior night, petitioner felt much better. An MRI of her brain was normal. On examination, petitioner continued to stutter when she spoke but talked normally if she whispered. She did not have any focal deficits. She was able to get off the bed and walk normally backwards, but when she walked forward, she buckled and seemed jerky, but did not fall or hit herself. She managed to turn around and sit on the bed. Dr. Urrutia's assessment was that petitioner had "symptoms that do not fit a physiologic paradigm." Id. Her symptoms might be a reaction due to anxiety. He suggested petitioner see a therapist. He prescribed clonazepam[37] for management of her symptoms as they were likely related to anxiety and because she felt better after taking Ativan. His impression was speech dysfunction and gait dysfunction. Id. On the same day, petitioner was discharged.

On October 4, 2009, Dr. Christopher B. Oakley wrote the discharge summary. Med. recs. Ex. 55, at 6. Under the impression that petitioner received flu vaccine on August 30, 2009,

[35] Astasia-abasia is "motor incoordination with an inability to stand or walk despite normal ability to move the lower limbs when sitting or lying down, a form of hysterical ataxia." Dorland's at 167. Ataxia is "failure of muscular coordination; irregularity of muscular action." Id. at 170. Hysterical ataxia is "ataxia that is part of a conversion disorder." Id. at 171.

[36] Ativan is "trademark for preparations of lorazepam." Dorland's at 173. Lorazepam is "a benzodiazepine with anxiolytic and sedative effects, administered orally in the treatment of anxiety disorders and short-term relief of anxiety symptoms and as a sedative-hypnotic agent." Id. at 1074.

[37] Clonazepam is "a benzodiazepine used as an anticonvulsant in the treatment of Lennox-Gastaut syndrome and of atonic and myoclonic seizures and as an antipanic agent in the treatment of panic disorders." Dorland's at 373.

Dr. Oakley wrote that petitioner developed URI symptoms that progressed to severe fatigue and muscles aches, but she continued to work. Petitioner told Dr. Oakley that she was told she had lupus and Lyme disease. MRIs of petitioner's cervical, thoracic, and lumbar spine were normal as was a brain MRI. After receiving Valium,[38] she improved. With no other intervention, her symptoms improved and she was discharged on clonazepam. Her symptoms were thought to be a stress reaction to what happened weeks earlier when her liver enzymes and CPK were elevated. Her symptoms might be an anxiety reaction, which was explained to her. She was advised to undergo physical therapy and have psychological support with a therapist or psychiatrist. "She was also informed that the relationship with the flu vaccine was not clear." Id. She was expected to improve on clonazepam, positive encouragement, and time. Id. Petitioner was able to speak without abnormalities in a soft whisper with some mild stuttering that improved with encouragement. Id. at 7. Her eyes did not have nystagmus. On motor examination, she had normal bulk and tone. Her strength was 5 out of 5 in all extremities. Her reflexes were 2+/brisk with no clonus and downgoing toes. Finger-nose-finger was slow and brought out severe vertical head tremor as did heel-to-shin-testing. She was able to stand without assistance. Her gait was narrow-based. After taking two to three steps, she began to have severe shaking of her head in a vertical motion, then began to have a bouncing motion of her legs as if her knees were going to give way, but she did not fall. Her gait improved with reassurance and encouragement.

On October 6, 2009, petitioner visited her PCP Dr. Ho. Med. recs. Ex. 22, at 12. Petitioner reported that when she touched her anterior left thigh or fastened a belt around her left thigh, she could walk completely normally. Id. When she did not do this, she could only run forward, but she could walk backward and sideways normally. Id. She also reported that her difficulty with talking was relieved by placing a hand on her chin. Id. Petitioner also claimed that when she took her husband's 5mg Valium tabs, she was "perfectly normal" for about 10 hours. Id. She had plans to see a dystonia specialist at Mayo Clinic in November 2009. Id. Upon physical examination, Dr. Ho noted that petitioner was well-nourished and well-groomed, and had normal strength and tone overall with no atrophy, spasticity or tremors while seated. Id. at 14. Dr. Ho suggested that if further extensive workups were unrevealing, he would strongly consider a conversion disorder, delusional disorder, or other psychogenic etiology. Id. at 14.

On October 7, 2009, petitioner completed a Vaccine Adverse Event Reporting System ("VAERS") form, giving the wrong date of vaccination, August 30, 2009, and stating onset was on September 3, 2009 at 6:20 a.m. Med. recs. Ex. 3, at 2. She described the adverse event symptoms as sore throat, nasal congestions, followed by fever, body aches, chills, and headache. She states she was hospitalized for eight days and her reaction resulted in permanent disability. She states that three days after her flu vaccination, on September 3, 2009 (which was 10 days after her flu vaccination on August 23, 2009), she came down with flu-like symptoms. On September 12, 2009, she began fainting and going into violent convulsions. Two weeks later, her legs began to fatigue and her neck began to shake periodically. On September 23, 2009, she lost the ability to walk and walked as if she had MS. She could not walk straight and her entire

[38] Valium is "trademark for preparations of diazepam." Dorland's at 2020. Diazepam is "a benzodiazepine used as an anti-anxiety agent in the treatment of anxiety disorders and for short-term relief of anxiety symptoms, … also as a skeletal muscle relaxant, anticonvulsant, antitremor agent, antipanic agent . . . ." Id. at 512.

body shook when she walked. Id. On September 24, 2009, she lost the ability to talk (stuttering) and had neck pulls and strains when talking. Her symptoms continued and she was diagnosed with dystonia. Id. at 3.

On October 9, 2009, petitioner saw Dr. Christine M. Cosgrave, a psychologist, for her cognitive and emotional issues.[39] Med. recs. Ex. 45, at 1-3. Petitioner displayed significant physical and neurological difficulties and Dr. Cosgrave recommended that petitioner seek an evaluation by a neurologist and neuropsychologist. Id. at 2. Dr. Cosgrave referred petitioner to Dr. Sidney W. Binks III, a clinical neuropsychologist, for a neuropsychological evaluation. Id.

On October 12, 2009, petitioner saw physical therapist Dallas A. Simons, a visit that Ms. Simons described in a letter entitled "To Whom It May Concern," dated May 12, 2010. Med. recs. Ex. 16, at 1. Petitioner told PT Simons that she had a muscular problem, which she felt was a complication of flu vaccination. On October 12, 2009, PT Simons evaluated petitioner's gait and found petitioner could walk backward fairly normally, but was very ataxic walking forward. If petitioner held her thigh, she could walk much more normally. Petitioner spoke very softly, but if she held her chin, her volume and cadence improved. PT Simons felt this was beyond her competency to evaluate and did not complete a formal evaluation. PT Simons gave petitioner the name and phone number of a physical therapist more experienced in neurological problems and dystonia and did not provide petitioner with treatment. Id.

On October 13, 2009, petitioner saw Dr. Ho, requesting a medical letter supporting extension of her disability benefits. Med. recs. Ex. 22, at 9. She said that when she touched her anterior left thigh or fastened a belt around her left thigh, she could walk completely normally. When she did not do this, she could only run forward, not walk, but she could walk backward and sideways normally. Her difficulty talking was eased by placing a hand on her chin. She was discharged from Johns Hopkins on Klonopin[40] but, after her first dose, she had "convulsions." Id. When she took one of her husband's Valium tablets, she was perfectly normal for about 10 hours. Id. Dr. Ho discussed petitioner's findings in her rather extensive work up and said if further extensive workups at academic centers were unrevealing, he "would strongly consider a conversion d/o [disorder], delusional d/o, or other psychogenic etiology." Id. at 10 and 11. Dr. Ho performed a physical examination and found her reflexes normal, but her gait broad-based "(VERY ERRATIC [Dr. Ho's emphasis])", spastic, stamping (sensory ataxia) and waddling. Id. at 11. Dr. Ho provided a letter to excuse petitioner from work or school due to her "undergoing work up and treatment for dystonia of uncertain etiology." Id. at 38.

[39] Petitioner first met Dr. Christine M. Cosgrave on April 10, 2009, when she accompanied her then-husband, B.J., for an initial psychotherapy intake session for him. Med. recs. Ex 187, at 3. However, by referral from her physician, petitioner began her own individual psychotherapy with Dr. Cosgrave on October 9, 2009 to obtain cognitive and emotional help "associated with possible reaction to an influenza vaccine she reportedly received in September 2009." Id. Subsequently, petitioner obtained services from Dr. Cosgrave on October 9, 2009, December 7, 2009, February 12, 2010, and March 5, 2010. Id.

[40] Klonopin is "trademark for a preparation of clonazepam." Dorland's at 989. For the definition of Clonazepam, see supra, n.33.

On October 15, 2009, petitioner and her then-husband saw Dr. Ruben Cintron,[41] a neurologist. Med. recs. Ex. 1, at 10. The history was that within a few weeks of flu vaccination in August, she developed flu-like and numerous neurological symptoms, including sensory symptoms. She had a fair amount of difficulty with her motor system, i.e., speech, gait, movements, tolerance to eating, and syncope, which had been labeled as a dystonic reaction or disorder related to vaccination. Id. She was initially diagnosed with rhabdomyolysis with a CPK in the 13,000 range and elevation of her liver enzymes. MRI of the brain, spinal fluid analysis, and multiple serologies were all unremarkable. She could not walk forward or sideways,[42] only backward. When she walks, she has jerking episodes. Id. During physical examination, petitioner was alert and oriented. Her speech was interrupted and non-fluid, although much better when whispering or singing. Id. at 10. She did not have visual dysmorphism or dystonic features. Id. at 10-11. When she tried to exert power, she had a fair amount of clinical rhythmic jerking throughout her shoulder girdles. Id. at 11. At least once, petitioner lost control with an episode of jerking that seemed to take over her ability to function motorally. During that time, her heart rate did not seem to have changed, she was aware, and she came back quickly without lying down. When petitioner tried to walk forward, her legs collapsed. When she walked backward, she did better. Her reflexes were symmetrical with downgoing toes. She had normal CSF, brain MRI, test results for Lyme disease and other serologies. Her CPK elevation ranged from 13,000 to 300. Id. Dr. Cintron's impression was that petitioner's history did "suggest vaccination induced motor disorder, with some dystonic and myoclonic features." Id. Dr. Citron did not believe that petitioner had a functional disorder based on her behavior and the consistency of the behavior. Id. Dr. Cintron prescribed Cogentin[43] and gave petitioner and her then-husband information on plasmapheresis[44] and intravenous immunoglobulin ("IVIG"). Id.

From October 19-22, 2009 and October 26-28, 2009, and on November 18, 2009, and December 1, 2009, petitioner had treatments with Dr. Rashid Buttar, D.O.[45] at his Center for

[41] Dr. Cintron received his MD at Wake Forest School of Medicine. He did an internship in internal medicine at Georgetown University Hospital, followed by a residency there in neurology. He did a fellowship in Neuromuscular Diseases and Electrodiagnostic Medicine at George Washington University Medical Center. In 1995-1996, Dr. Cintron became board certified in Adult Neurology and Neuromuscular /EMG. He considers himself a general neurologist with special interest in neuromuscular disorders/EMG, migraines, movement disorders and memory disorders. Physicians and Practitioners, NEUROSCIENCE CONSULTANTS, PLC, www.nscpic.com/ruben-cintron-m-d/ (last visited Jan. 9, 2019).

[42] This is the first medical record to note petitioner could not walk sideways. In previous histories, she said she could walk sideways and backward.

[43] Cogentin is "trademark for preparations of benztropine mesylate." Dorland's at 382. Benztropine mesylate is "an antidyskinetic believed to act by partially blocking central cholinergic receptors, so that cholinergic and dopaminergic activity in the basal ganglia is more balanced; used in the treatment of parkinsonism . . . ." Id. at 209.

[44] Plasmapheresis is "the removal of plasma from withdrawn blood, with retransfusion of the formed elements into the donor; generally, type-specific fresh frozen plasma or albumin is used to replace the withdrawn plasma." Dorland's at 1456.

[45] The North Carolina Board of Medical Examiners issued a reprimand to Dr. Buttar on March 26, 2010 for not informing his patients about the types of treatment and therapies he was recommending to them. Dr. Buttar consented to the reprimand. Licensee Information: Rashad Ali Buttar- DO Full and Unrestricted, NORTH CAROLINA MEDICAL BOARD, https://wwwapps.ncmedboard.org/Clients/NCBOM/Public/LicenseeInformation/Details.aspx?EntityID=66840&Pub

Advanced Medicine & Clinical Research in Huntersville, North Carolina. See med. recs. Ex. 59, at 59-83. Petitioner arrived at Dr. Buttar's clinic with her then-husband, brother-in-law, Stan Kurtz,[46] and a camera crew on October 19, 2009. Id. at 80. Petitioner heard about Dr. Buttar[47] from Generation Rescue. Id. at 304.

Petitioner gave Dr. Buttar a timeline including her impression she received flu vaccine on August 30, 2009. Id. at 90. She described waking up on September 3, 2009 with a sore throat and congestion. That afternoon, she started feeling fatigue and was very hot. She took a decongestant and two Aleve. After dinner, she started feeling hot and nauseated. She lay on the couch and had body aches all over, most severely at the hip muscle and biceps where she had been working out. From September 4-7, 2009, she mostly slept, had no appetite, and was very lethargic. Her sore throat and congestion continued. Id. From September 8-11, 2009, she went back to work, but was constantly lethargic and fatigued. She had a mild sore throat in the morning, continuing congestion, and a green ball of mucus in the morning. In the evening of September 11th, she drank a glass of wine and two mixed drinks over a four-hour period starting at 7:00 p.m. At 11:00 p.m., she started vomiting violently and uncontrollably. On September 12th, she woke up very weak, dehydrated, and tired. She drank lots of fluids and ate breakfast. Around 11:00 a.m., she became very lightheaded and hot, but her hands and feet were cold. Id. She fainted while sitting on the couch. She lost all muscle control while trying to walk. She states she began convulsing and hyperventilating in the car. Id. From September 18-20, 2009, petitioner states she was still lethargic and weak. She "finally figured out I was fainting and going into convulsions after eating." Id. Petitioner would eat while lying down to avoid fainting. She still was congested with greenish yellow mucus each morning. Id. Petitioner notes that even by September 28, 2009, she still had congestion with yellow and white mucus. Id. at 95.

Dr. Buttar stated that petitioner presented to his clinic "in emergent state, in severe distress with labored breathing, experiencing multiple witnessed, back to back seizures or contractures, extraordinary gross motor deficits, loss of coordination, absence of fine motor skills, inability to articulate with dysphonia, and unable to ambulate." Id. at 74. Petitioner reported that she had done an 8K race two days previously. Id. Petitioner claimed that she was able to breathe better while running than at rest. Id. Petitioner lost ten pounds over the last five

licFile=1 (last updated Jan. 16, 2019). On February 10, 2011, the Hawaii Medical Board denied Dr. Buttar's application for medical licensure due to the NC Board action. Id. Dr. Buttar lists his area of practice as occupational and environmental medicine. Id. He was licensed in Texas until 1994. Id. Dr. Buttar does not have any hospital affiliations. Health, U.S. NEWS & WORLD REPORT, https://health.usnews.com/doctors/rashid-buttar-626895 (last visited Jan. 19, 2019).

[46] Mr. Kurtz has invented treatments to cure autism and was awarded top 20 "Hall of Fame" status by Generation Rescue. www.stankurtz.org/about/affiliations.html (last visited Jan. 10, 2019). Generation Rescue is an organization promoting treatment for autism. www.generationrescue.org/who-we-are/ (last visited Jan. 10, 2019).

[47] Dr. Buttar has a website: https://www.drbuttar.com (last visited Feb. 12, 2019). It states on its home page that The Center for Advanced Medicine and Clinical Research specializes in addressing the needs of patients suffering from chronic disease, treatment failures, difficult to diagnose conditions, cancer, autism, cardiovascular disease, neurodegenerative disease, environmental toxicity, heavy metal toxicity, chemical toxicity, and metabolism disorders. It also states its treatments are "so effective that even the North Carolina Medical Board is trying to suppress the truth" and cites to another website: DrButtarTruth.org. Id.

to seven days and experienced inability to "keep foods down." Id. Dr. Buttar noted that petitioner experienced nausea, vomiting, and dry heaving throughout the day. Id. at 80. Dr. Buttar diagnosed petitioner with dystonia by history, acute respiratory distress; acute viral, post-immunization encephalopathy; allergic reaction to medicinal substance; and rule out heavy metal toxicity. Id. at 75.

During the following days after arriving at Dr. Buttar's office on October 19, 2009, petitioner received IV chelation therapy to "have ability to speak and walk." Id. at 68-80. Dr. Buttar noted on October 19, 2009 that petitioner tolerated the IV well and it made a "huge difference." Id. at 70-71. In his note of October 20, 2009, Dr. Buttar stated petitioner was "ecstatic" and the nursing staff was "celebrating" since petitioner was able to walk into the IV suite and talk in a normal voice. Id. at 69. On October 21, 2009, petitioner received IV treatment and hyperbaric oxygen therapy ("HBOT"). Id. at 79. On the same day, petitioner called Dr. Buttar with complaints of seizing again and her voice going out. Id. at 67. Dr. Buttar visited petitioner and gave her TD-DMPS,[48] and eventually petitioner stated that she felt good. Id.

On October 22, 2009, petitioner continued to have the ability to speak and walk and underwent a quantitative electroencephalogram ("qEEG"). Id. Myra A. Preston,[49] Ph.D., evaluated petitioner's qEEG data and concluded petitioner's left parietal and temporal regions contained abnormal activity that should be further investigated. Id. at 87-89. During the time that Ms. Preston thought petitioner was having a seizure, the qEEG recording consisted of muscle artifact as petitioner had strong muscle contractions in her facial muscles. Id. at 88. Then the "seizure" subsided and the qEEG returned to baseline. Petitioner applied a cream to her forearms and the qEEG data began to appear more normal in the parietal and temporal

[48] TD-DMPS stands for transdermal 2,3-Dimercaptopropane-1-sulfonate, which "is a metal chelator approved in Europe for oral or intravenous use for heavy metal poisoning. Transdermally applied DMPS (TD-DMPS) is used by some alternative practitioners to treat autism, despite the absence of evidence for its efficacy." Plasma and urine dimercaptopropoanesulfonate concentrations after dermal application of transdermal DMPS (TD-DMPS), 9 J MED TOXICOL 9-15, at 9 (2013).

[49] Ms. Preston has a website called www.siberimaging.com in which she states that she offers through qEEG and neurofeedback enhancement of central nervous system functioning to promote peak performance, intelligence, alertness, focus, strength, balance, positive mood, and awareness. She claims she and Kim Phillips, Clinical Director, developed and patented this in 1993. SIBER IMAGING, WWW.SIBERIMAGING.COM (last visited Jan. 11, 2019). Ms. Preston claims success in training individuals with the following disorders: alcohol & substance abuse, medical reduction & withdrawal, depression, post-traumatic stress disorder, panic disorder, obsessive compulsive disorder, bipolar disorder, dementia, eating disorders, attention deficit disorder, attention deficit hyperactivity disorder, adult attention deficit disorder, chronic fatigue syndrome/myalgic encephalomyelitis, various sleeping disorders, fibromyalgia, post-viral syndromes, premenstrual stress syndrome, migraine headache, hypertension, gastrointestinal dysfunction, bowel dysfunction, bladder dysfunction, heart rate regulation, chronic pain syndrome, vulvodynia pain, dysautonomia, epilepsy seizure disorders, Tourette's syndrome, tic disorder, autism, autism spectrum disorders, brain injury, closed head injury, spinal cord injury, anoxic brain injury, and stroke. Id. Chronic Fatigue Immune Dysfunction Syndrome ("CFIDS") was the focus of Ms. Preston's Ph.D. dissertation. Id. She claims that the reasons CFIDS clients have normal EEG results on conventional EEGs is that they are done with the client's eyes closed. Ms. Preston does qEEGs on CFIDS clients with their eyes open, and gets an abnormal response showing the clients have metabolic encephalopathy. Id. Ms. Preston received her doctorate in Psychophysiology in 1994 from The Union Institute. Id. The North Carolina Medical Board accused Ms. Preston of practicing medicine without a license through her company Siber Imaging, and selling a BrainMaster "brain mapping machine." Medicine, COURTHOUSE NEWS SERVICE (Feb. 22, 2011), http://www.courthousenews.com/medicine-2.

regions for 10 to 15 minutes. However, neither region became completely normal. Ms. Preston did not see any additional seizures and petitioner began to speak normally. Id. During the next 21 minutes, petitioner applied more of the cream and the qEEG maintained improvements. Id. Ms. Preston characterized the one seizure as an absence seizure. Id. In addition, Ms. Preston added that, in her experience, "the data acquired from the left temporal and parietal regions most resemble data correlated with myoclonic seizures," and went on to note that medical literature identifies these types of findings with acute insults. Id. at 89. However, Ms. Preston also noted that "this evaluation should not be considered diagnostic." Id. at 87.

While seeking treatment at Dr. Buttar's office, petitioner's urine tests revealed high levels of mercury, nickel, copper, lead, zinc, and manganese. Id. at 16-40. Petitioner's urine test revealed low levels of molybdenum. Id. at 42.

On October 30, 2009, petitioner visited Dr. Cintron for a neurological follow-up. Med. recs. Ex. 1, at 13. She reported that she had been working in Charlotte with chelating therapy and felt that she was making progress. Dr. Cintron thought petitioner had a minor seizure in his waiting area but by using a compound that Dr. Buttar gave her, she dropped into her forearm and was able to calm down. Her speech seemed somewhat easier to understand. Id. Dr. Cintron also ordered an EEG, the result of which was normal. Id.

On December 7, 2009, petitioner saw Dr. Cosgrave again and her gait and speech had "somewhat improved." Med. recs. 45, at 2.

Also, on December 7, 2009, petitioner visited Dr. Randolph R. Stephenson, a neurologist. Med. recs. Ex. 22, at 36-37. Petitioner stated that in September, she was hospitalized for rhabdomyolysis following either a viral illness or flu vaccination. Afterward, she started developing problems with walking and speech. She felt as though she had difficulty moving her legs while walking, but "remarkably," she had no problems running. Id. at 36. She had problems with stuttering. She said numerous doctors suggested she might be having a stress reaction and suggested she see a neuropsychologist. She was going to North Carolina for chelation therapy, "although it is unclear what is being chelated." Id. She reported that she was having frequent "seizures" at the rate of around 60 per day, during which she would shake frequently. She denied losing consciousness or having urinary incontinence during these episodes. She did not appear to be having these episodes anymore at the same frequency. Id. at 36. Petitioner also reported that she continued to exercise regularly, but after running she sometimes passed out. Id.

On physical examination, petitioner's blood pressure was 166/86. Id. at 37. Dr. Stephenson noted petitioner had normal cognition, comprehension, and vocabulary, but frequently talked in a British or Australian accent. Id. Petitioner said it was part of the changes in her speech. Id. She also had frequent stuttering. Petitioner had normal muscle bulk, strength, and tone. Id. When Dr. Stephenson asked her to point or hold or write something, petitioner would start shaking her head and hands vigorously. The tremor would be in several planes of direction and was highly distractible. There was also a highly suggestible component to it so that Dr. Stephenson could easily bring out the tremor just by having petitioner talk about it. She had normal light touch, pinprick, temperature, and vibratory sensation. Her reflexes were 1 to 4 and

symmetric at the biceps, triceps, patellae, and ankles. Her toes were downgoing. Petitioner had a "very clear" astasia-abasia gait when walking. Id. She would often appear off balance without actually falling. When Dr. Stephenson watched her closely, he saw clearly that her balance was actually better than average given some of the positioning her body took while walking. Id. Dr. Stephenson's stated he was not entirely sure what caused all of petitioner's movement complaints. However, he wrote she had "a very clear functional component to the majority of her exam. There were no neurological findings that would suggest any particular organic disease process." Id.

On December 11, 2009, petitioner saw Dr. Sidney W. Binks, III, Ph.D., at the recommendation of Dr. Christine Cosgrove, the clinical psychologist. Med. recs. Ex. 19, at 1. Dr. Binks is a neuropsychologist and clinical psychologist. Id. While giving a history to Dr. Binks, petitioner said that her MRI had been painful as if she were on fire. Id. at 2. She said that looking at carpet patterns could cause her whole body to shake and a speech problem. She said she was having 60 seizures a day. She reported great difficulty with fine motor coordination. Her mental health history was positive for bulimia at age 13 for one year. She was currently in psychotherapy with Dr. Cosgrove. Her sister had attempted suicide. Dr. Binks interviewed petitioner on December 11, 2009 and evaluated her on December 19, 2009 and January 16, 2010 for about seven hours. Petitioner drove herself to and from the testing on both days. She did not have any muscle contractions or involuntary movement as she entered or exited Dr. Binks' office on either testing day. However, on the day of the interview, she walked extremely awkwardly. She could not walk forward but instead walked sideways. Dr. Binks interviewed her when she was with her then-husband who often reported her history because she had difficulty speaking and recalling. Id.

Petitioner frequently became frustrated during testing when she was asked to pronounce words. She had muscle contractions and seemed to put great effort into producing the answer. She also had a stutter. Her arm muscles and face muscles would simultaneously contract during these times. Her right-sided muscle movement was more pronounced, including her arm and face. The more frustrated she became, the more the muscle movements increased. Dr. Binks did not observe these muscle contractions during general conversations. Her general speech had somewhat of a British accent. Her volume would occasionally be high-pitched. Id.

Petitioner reported she had difficulty focusing on a page when asked to perform certain tasks such as sentence comprehension, and she used a piece of blank paper with a small cut in the middle to read line by line. She would spell a word out loud as she wrote it on the paper because she indicated that made it easier. She said attempting to look at pages gave her a headache. Id. Petitioner had great difficulty pressing her finger down on a button and had muscle contraction increase in the finger tapping test. Id. at 3. Petitioner reported that some of her physical complaints had lessened, but her speech and cognitive symptoms had recently worsened. Id.

Dr. Binks also conducted a personality/psychopathology test in addition to the interview. Id. Petitioner's validity scales suggested that she was "uncomfortable acknowledging personal faults" and presented herself in an extremely positive light by denying many minor faults and

shortcomings that most people acknowledge." Id. Dr. Binks noted that "this level of virtuous self-presentation is uncommon and likely resulted in an underestimate of personality-related clinical findings." Id. Dr. Binks stated that due to petitioner's preoccupation with physical health concerns, "she is likely prone to developing physical symptoms in response to stress." Id. at 4. Dr. Binks concluded, "Problems with her emotional life, thought process and behavior could not be ruled out given the tendency to under-report even normal psychological imperfections." Id. Dr. Binks' impression was that petitioner had weakened, but not impaired, cognitive skills. Id. Dr. Binks believed that intensive speech and language evaluation/therapy should be pursued, working on lowering petitioner's expectations to realistic levels could be helpful, and stress reduction techniques would be useful. Id. Although petitioner's cognitive skills were currently weakened, Dr. Binks did not consider them impaired but at functional levels. He suggested psychotherapy to help lower her perfectionism to realistic levels. Dr. Binks noted petitioner was under tremendous stress and he suggested stress reduction. He stated, "She would do well to avoid the pull toward being a public spokesperson for others and instead focus on her own wellbeing and rehabilitation. It is clear that stress exacerbates her symptoms." Id.

On December 18, 2009, petitioner saw Dr. Cintron for a neurological follow-up. Med. recs. Ex. 1, at 14. Petitioner's gait and speech were better, but she still had difficulty walking unless she walked sideways, and her speech tone changed during her visit. The EEG[50] done in Charlotte implied petitioner had epilepsy, but Dr. Cintron did an EEG himself and it was normal. He looked at the Charlotte EEG with petitioner and did not see any abnormalities. Petitioner "alleged" [Dr. Cintron's verb] that her heart beat went to almost 200 when she exercised and Dr. Cintron told her to stop exercising until she saw a cardiologist. He states, "Ultimately, this appears to be a very complicated situation with post vaccination disorder. . . ." Id.

On December 18, 2009, petitioner saw Dr. Mark P. Tanenbaum at the Cardiovascular Group, P.C., for a cardiology assessment for her increased heart beat while running. Med. recs. Ex. 13, at 7. Petitioner said she received flu vaccine on August 23, 2009 and, within a couple of weeks, she developed apparent flu-like symptoms, neurologic symptoms, and sensory symptoms. She had some motor, gait, and speech abnormalities. She had an episode including syncope on September 12th after eating. She was admitted to Loudoun Hospital which diagnosed her with rhabdomyolysis. She was discharged on September 14th but readmitted on September 17th with recurrent syncope. She was discharged, but she was readmitted with similar symptoms at Fairfax Hospital on September 26th. She said she was told she may have GBS related to her flu vaccination. She went to Johns Hopkins University Hospital and was treated with Ativan and Klonopin. She had an EEG which was normal. She went to Charlotte, NC, for chelation therapy to remove apparent excess mercury. She had been usually quite active, running three miles in 25 minutes. During the last several months, when she ran, her heart rate would increase more quickly up to 180 beats a minute associated with fatigue. Id. In a review of systems, Dr. Tanenbaum wrote petitioner denied any difficulty speaking. On physical examination, petitioner did not have any gross motor or sensory deficits. Id. at 8. Dr. Tanenbaum's impression was

[50] This must be the qEEG that Ms. Preston did since there is no other EEG that petitioner underwent while staying in North Carolina to receive evaluation and treatment from Dr. Buttar.

"apparent vaccination-induced motor disorder, along with apparent dystonic and myoclonic features." Id. Dr. Tanenbaum suspected that petitioner's elevated heart rate represented sinus tachycardia, which "might be exacerbated by her ongoing problem with apparent vaccination-induced motor disorder, along with apparent dystonic and myoclonic features." Id. at 8.

On December 21, 2009, petitioner had a stress echocardiogram. Id. at 11. Dr. Nick Cossa concluded petitioner had a normal stress echocardiogram without evidence of ischemia. She had a normal heart rate and blood pressure response to exercise, normal exercise capacity, no chest pain, ischemic EKG changes, or left ventricular wall motion abnormalities with exercise. She had a transient syncope after exercise secondary to neurocardiogenic hypotension. Id.

On December 28, 2009, petitioner went to a follow up appointment with Dr. Cintron. Med. recs. Ex. 1, at 15. He thought petitioner walked better and her speech was more fluid. She still had an "accent" [quotation marks are by Dr. Cintron]. She sounded dystonic and still had some occasional syncopal episodes although her blood pressure in Dr. Cintron's office was fine. Apparently, she had a syncopal episode while on a treadmill in the cardiologist's office, but the office felt she did not have arrhythmia. Petitioner found that Valium allowed her to run better and she took it sporadically. She still had better heat intolerance to some extent, and she was better with not being overwhelmed in a stimulating environment. Dr. Cintron concluded that petitioner's problem was vaccine related consisting of dystonia and possibly some autonomic problem. Id.

On January 19, 2010, petitioner saw Dr. Cintron. Med. recs. Ex. 1, at 9. She seemed better. She had learned that with pain in her left quadriceps, she was able to walk better because she got distracted. She also learned that by looking to the left, she could walk straighter. Petitioner's speech was significantly better that day. Dr. Cintron thought petitioner had some type of dystonia related to her insult and he did not believe her situation was functional, "although certainly there is some bizarre aspects to her disease." Id.

On February 5, 2010, Dr. Cintron had petitioner undergo a Transcranial Doppler because of her vertigo/dizziness and vertebrobasilar[51] syndrome. Med. recs. Ex. 1, at 22. The result was normal. He also checked her carotid arteries for stenosis[52] because of transient ischemic attacks. Id. at 23. The result was no significant stenosis in the right internal carotid artery and minimal 1-15% stenosis in her left internal carotid artery. Both external carotid arteries had no significant stenosis. Id.

On February 5, 2010, petitioner went to the Reston Hospital ED, complaining of vomiting. Med. recs. Ex. 50, at 5. This started the day before. Id. at 6. Petitioner said she felt dizzy on standing and had increased thirst. On physical examination, her motor exam was normal in all extremities and her sensory exam intact. She had no abnormalities on the cerebellar exam. Her cardiac rate and rhythm were normal. Her extremities were normal. Petitioner told Dr. David Kruse that she had autonomic dysfunction after a flu shot. Id.

[51] Vertebrobasilar pertains to vertebrae and arteries. Dorland's at 2051.
[52] Stenosis is "an abnormal narrowing." Dorland's at 1769.

Petitioner had driven herself to see Dr. Cintron that morning and "developed a syncopal episode with thickened speech with a British accent and inability to walk." Id. at 12. Her glucose was low at 73 when the normal range is 74-106 mg/DL. Id. at 19. Dr. Kruse diagnosed petitioner with clinical dehydration and discharged her. Id. at 7.

On February 8, 2010, petitioner went to Inova Fair Oaks Hospital Rehabilitation Center Speech-Language Pathology (SLP) for an evaluation, to which Dr. Cintron sent her. Med. recs. Ex. 17, at 7. The onset of petitioner's speech difficulty was noted as September 3, 2009. The history was that petitioner developed an adverse reaction to a flu vaccination, and was hospitalized for seizures and decreased ambulation. She also developed vasodepressor syncope and reported a decrease in speech manifesting as stuttering, foreign accent, and blocks. She stated she had a decrease in doing simple math and in memory. Id. The assessment was moderately severe cognitive, language processing/mild speech production disorders. Processing deficits were noted for combined modalities. She had significant breakdowns in attention skills, including selective and divided. This impacted her ability to complete basic routines of activities of daily living ("ADLs"). When petitioner's cognition, processing, and attention are challenged, this impacted her verbal expression complicated by dysfluencies/blocks. Id. The pathologist (whose signature is illegible) wrote petitioner would benefit from skilled speech therapy intervention to improve her cognitive skills for ADLs. Id. As part of petitioner's evaluation, the therapist noted petitioner spoke English with an Australian accent and when stressed, her speech became extremely dysarthric with spasmodic head movements which the therapist diagnosed as transient neurogenic stuttering brought on by stress, cognitive stress vs. distraction and auditory distraction. Id. at 8. During more testing on February 15, 2010, the therapist noted that petitioner was able to jog in place to "refresh" herself with more normalized speech afterwards. Id. at 9. On March 15, 2010, the therapist noted that a low dosage of Valium appeared to facilitate petitioner's ability to tolerate multi-modal stimulation and function more optimally. Id. at 5.

On February 12, 2010, Dr. Cintron sent petitioner to Cardiovascular Group again. Petitioner saw Dr. Pradeep Nayak. Med. recs. Ex. 13, at 4; Ex. 53, at 66. Dr. Nayak diagnosed petitioner with vasodepressor[53] syncope, demonstrated following her stress echocardiogram about six weeks previously. Med. recs. Ex. 12, at 5. He also diagnosed her with "apparent vaccination-induced motor disorder," elevated heart rate early in exercise, mild rhabdomyolysis in 2009, and a leg injury with desire to return to running. Id. He recommended she see Dr. Walter L. Atiga.

On February 23, 2010, petitioner saw Dr. Cintron. Med. recs. Ex. 1, at 8. Her speech was mildly impaired with mild dystonic-type filter. Dr. Cintron found that her neuropsychological testing was somewhat confusing in his interpretation. He thought it interesting that when petitioner exercised, she did better.

[53] Vasodepression is a "decrease in vascular resistance with hypotension." Dorland's at 2027.

On February 26, 2010, Dr. Cosgrave, petitioner's psychologist, mailed a letter to petitioner's former attorney. Ex. 11, at 1, and Ex. 45, at 2. She states that the extent of petitioner's impairments was beyond her scope of expertise. Id.

On March 2, 2010, petitioner underwent a brain MRI for foreign accent syndrome.[54] Med. recs. Ex. 53, at 68. Dr. Arun Kumar compared the results with petitioner's brain MRI, done October 3, 2009. Petitioner's ventricles, sulci, and cisterns were normal for her age. She did not have hydrocephalus, abnormal signal intensity in the brain parenchyma, hemorrhage, mass, mass effect, or midline shift. She did not have evidence of restricted diffusion to suggest an acute or subacute infarction. Her mesial temporal lobe structures were symmetric. She did not have evidence of cortical dysplasia. Her flow voids were maintained. There was mucosal inflammation in the mastoid sinuses, ethmoidal air cells, and frontal sinuses. Her orbits were normal. Her mastoid air cells were clear. Dr. Kumar's impression was petitioner did not have an acute intracranial process and she had mild sinus disease. Id.

On March 3, 2010, petitioner underwent a F-18 FDG PET/[55]CT scan of her brain for cognitive impairment and foreign accent syndrome. Id. at 69. Dr. Stuart A. Fruman compared the results with petitioner's brain MRI, done on October 2, 2010. Petitioner had symmetric distribution of FDG metabolism through her brain parenchyma. She did not have any abnormal areas of increased or decreased isotope accumulation. Dr. Fruman wrote petitioner had a normal brain PET/CT scan. Id.

On March 16, 2010, petitioner and her then-husband saw Dr. Cintron. Med. recs. Ex. 1, at 7. Petitioner's PET scan was normal. He told petitioner and her then-husband that he had not seen any evidence of any irreversible changes to any part of her central nervous system and he felt confident she would recover. At this point, he empirically labeled petitioner as having dystonic-type illness with other "not very well explained features and possible autonomic dysfunction secondary to her vaccination." Id. She seemed to have an exaggerated vasovagal syndrome because she faints after eating and exercising. Dr. Cintron states, "From the neurological perspective the symptoms are still bizarre, she has an accent which fluctuates, she is able to walk sideways but not forward, but no objective evidence on exam of where the problems are coming from." Id.

On March 23, 2010, petitioner saw Dr. Atiga, a cardiologist, for an evaluation and management of syncope. Med. recs. Ex. 13, at 1. Dr. Nayak referred petitioner to Dr. Atiga for

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[54] "Foreign accent syndrome (FAS) is speech disorder that causes a sudden change to speech so that a native speaker is perceived to speak with a 'foreign' accent. FAS is most often caused by damage to the brain caused by a stroke or traumatic brain injury. Other causes have also been reported including multiple sclerosis and conversion disorder and in some cases no clear cause has been identified." What is Foreign Accent Syndrome?, THE UNIVERSITY OF TEXAS AT DALLAS, https://www.utdallas.edu/research/FAS/ (last visited Jan. 25, 2019).

[55] Positron emission tomography (PET) is "tomography accomplished by detection of gamma rays emitted from tissues after administration of a natural biochemical substance (e.g., glucose, fatty acids) into which positron-emitting isotopes have been incorporated. The paths of the gamma rays, which result from collisions of positrons and electrons, are interpreted by a computer, and the resultant tomogram represents local concentrations of the isotope-containing substance." Dorland's at 1935. FDG is ^{18}fluorodeoxyglucose. Neil M. Davis ed., MEDICAL ABBREVIATIONS 144 (12th ed. 2005) [hereinafter "Med. Abbrev."].

management of vasovagal syncope. Petitioner stated she was healthy and active as a runner until she started having problems late summer 2009. She received flu vaccine for the third year in a row. A couple of weeks later, she started having recurrent episodes of syncope preceded by lightheadedness, warmth, and cold hands and feet. These would always occur soon after eating. The symptoms got so bad she would take a sip of water and then pass out, having nausea and vomiting afterwards. She went to Loudoun Hospital, and then Johns Hopkins, but was not able to get a clear and definitive diagnosis. She was featured on the local channel 5 news because she thought her symptoms were due to flu vaccine. Having read that news story, an organization that believes vaccines may cause autism contacted her because her symptoms could be consistent with mercury toxicity. As a result, she went to North Carolina and underwent chelation treatment because of elevated levels of mercury. The chelation therapy helped her feel better, but since it was prohibitively costly, she could not continue with it.

Presently, petitioner discovered that if she exercised, such as went running, she could eat without becoming nauseated, vomiting, or passing out. On days when she did not run, she had presyncope or syncope. She tore her left quadriceps and could not run. Since then, she has had a recurrence of symptoms so that she cannot eat without passing out. She is very sensitive to heat and, if she goes out on a warm day, she has lightheadedness, warmth, presyncope, syncope, nausea, and vomiting. Her then-husband hugged her tightly and compressed her neck at one point, and she passed out and had seizure-like activity. She had the same symptoms while undergoing a carotid Doppler examination. At her worst, she was unable to walk forward but could walk sideways or backward. However, when she began taking Neurontin[56] and after the dosage was increased, she was able to walk forward. Another "interesting" symptom was that she developed what sounds like a British accent although she is from Ohio. Id. at 1. She increased her salt intake by 2,000 mg of sodium and increased her fluids, but they did not have an effect. Id. at 1-2. Under job description, Dr. Atiga wrote professional cheerleader. Id. at 2.

Dr. Atiga's impression was that petitioner's symptom complex "does certainly time out with her receiving the flu vaccination." Id. Her symptoms were consistent with a vasovagal or neurocardiogenic mechanism. He writes, "It is interesting that on days when she has heavier physical exertion, her symptoms are improved enough that she can actually eat," but on days when she does not do this, her symptoms are worse. She also seemed to have evidence of carotid sinus hypersensitivity. Dr. Atiga writes, "I believe that she has an unusual form of neurocardiogenic syncope in that she has very heightened vagal tone and the reason that the exercise improves her on that day is because it increases her sympathetic tone." Id. To reduce petitioner's vagal tone, Dr. Atiga offered her either a Scopolamine[57] patch or Norpace.[58] Either

[56] Neurontin is "trademark for preparations of gabapentin." Dorland's at 1268. For a definition of gabapentin, see supra, n.8.

[57] Scopolamine is "an anticholinergic alkaloid, derived from several solanaceous plants, including *Atropa belladonna*, *Hyoscyamus niger*, *Datura* species, and *Scopolia* species. It has effects on the autonomic nervous system similar to those of atropine. It is used as an antiemetic, particularly in motion sickness." Dorland's at 1681.

[58] Norpace is "trademark for preparations of disopyramide phosphate." Dorland's at 1291. Disopyramide is "a cardiac depressant with anticholinergic properties, used as an antiarrhythmic." Id. at 547. An antiarrhythmic is "an agent that prevents or alleviates cardiac arrhythmia." Id. at 100.

drug would put her parasympathetic and sympathetic tone in more balance. He prescribed Norpace. Id.

On March 25, 2010, petitioner saw Dr. Cintron. Med. recs. Ex. 1, at 6. He states she is a very pleasant woman whom he follows for neurological implications secondary to exposure to a flu vaccine. She was doing significantly better since he increased her Neurontin to 200mg twice a day. She could walk forward. Her voice sounded much better. but she still had some difficulty eating unless she exercised. She was working with Dr. Atiga to help with her dysautonomia. Id.

Throughout February and April 2010, petitioner sought treatment at the Physical Medicine and Rehabilitation unit at Inova Fair Oaks Hospital. Med. recs. Ex. 53, at 41-59. Petitioner sought cognitive treatment that focused on processing multiple stimuli simultaneously and tuning out extraneous stimuli. Id. at 41. Through a hierarchy of graduated attention tasks and possibly a change in medications to help control her dystonia, Wendy Morgan, MS, CCC-SLP, wrote that petitioner had made significant gains in all cognitive areas. Petitioner was discharged because she moved out of the area. Id.

On May 21, 2010, petitioner saw Dr. Cintron. Med. recs. Ex. 1, at 5. Petitioner was trying to control her syncopal episodes that occurred when she was overheated. She seemed to be somewhat able to prevent them by exercising excessively as if she were trying to get her adrenaline going. Petitioner was seeing Dr. Atiga to see if she could get help with what appeared to be autonomic imbalance. From a neurological perspective, petitioner continued to have dystonia and speech difficulty, but had significantly improved over time. She was getting ready to move to California. Id.

On May 24, 2010, petitioner saw Dr. Farhad Zangeneh for an endocrine evaluation. Med. recs. Ex. 43, at 28. Petitioner was on disability and wanted Dr. Zangeneh to evaluate her adrenal gland, thyroid, and overall endocrine status. Id. A comprehensive metabolic test result was normal. Id. at 29. Dr. Zangeneh wrote that petitioner's blood pressure both sitting and standing was nearly identical, and she did not have orthostatic hypotension. Petitioner was on Depo-Provera. Dr. Zangeneh discussed with petitioner that, from an endocrine standpoint, he did not recommend Depo-Provera because of an increased risk for bone loss and for the development of metabolic syndrome. Id. On June 4, 2010, Dr. Zangeneh informed Dr. Ho that petitioner's lab results were normal and there was no endocrine etiology for petitioner's symptoms. Id. at 27.

On July 2, 2010, after petitioner moved to California, she saw Dr. Neil Q. Tran as her new PCP, at the Mission Internal Medicine Group. Med. recs. Ex. 42, at 28. In his note, Dr. Tran listed multiple problems including petitioner's intolerance to heat over 75^{o}F, anxiety, dystonia, autonomic dysfunction, dizziness, fatigue, diarrhea, and facial weakness. Id.

On July 2, 2010, petitioner began keeping a log of events and blood sugar readings. Med. recs. Ex. 42, at 26. She noted that on July 1, 2010, she last engaged in 30+ minutes of aerobics at noon. On Friday, July 2, 2010, she did not have any trouble all day eating or drinking. On Saturday, July 3, 2010, she became nauseated, dizzy, and unable to walk and talk after breakfast. This continued throughout the day at every meal. Later in the day, meals resulted in diarrhea.

On Sunday, July 4, 2010, she had very little appetite. Water now triggered her symptoms. Immediately after eating breakfast, she went into the hyperbaric chamber to stop her symptoms. She ate a peach and chicken with water. She became unable to walk or talk, became dizzy, had a headache, chest pain, and an upset stomach. She found the smell of food disgusting. She had an upset stomach upon drinking sips of water. She was dizzy when she got out of bed quickly. She had stomach pain. On Tuesday, July 6, 2010, she had a breakfast sandwich with apricots, a bagel with cream cheese and salmon, a pomegranate smoothie, and a small bowl of cereal with almond milk. She lost the ability to walk and talk, and had a painful headache upon simply sipping water if not allowed to lie down. She ate all her meals lying down. Her blood sugar was 100mg/dl before breakfast right after waking up, 117mg/dl ten minutes after eating breakfast, 203mg/dl two hours after eating, 92mg/dl at 2:51 p.m., and 82mg/dl at 7:29 p.m., which was two hours after eating. On Wednesday, July 7, 2010, she had a breakfast sandwich with apricots. She lost the ability to walk and talk, and had a headache when not able to lie down. She was extremely tired. She slept most of the morning. She felt she might be coming down with a cold because she had a sore/swollen throat, felt weak, and had a heavy chest for the prior couple of days. She had a lot more irritability with throbbing pain in her teeth. Her blood sugar was 82mg/dl before breakfast right after waking up, 99mg/dl two hours after eating, 125mg/dl at 4:45 p.m., and 97mg/dl before going to bed at 8:47 p.m. Id.

On July 12, 2010, petitioner was tested for levels of epinephrine and norepinephrine in her urine. The results were reported on July 16, 2010. Med. recs. Ex. 42, at 41. Her total was low at 15[59] when normal should be 26-121.

On July 15, 2010, Dr. Atiga conducted a tilt-table test. Med. recs. Ex. 23, at 1. Upon upright tilt, petitioner immediately developed slurred speech that progressively worsened over the next 15 minutes so that she could not speak at all. This occurred despite her having normal peripheral blood pressure and heart rate. She was then provoked with 0.4mg sublingual nitroglycerin and, shortly thereafter, she became dystonic, progressing to more severe dystonia, with tonic/clonic movements without loss of consciousness. Her blood pressure was 169/123 with a heart rate of 120 beats per minute. She was laid flat with her legs elevated. Petitioner had rapid resolution of dystonia, but her slurred speech persisted for nearly one hour. Id. Dr. Atiga wrote petitioner might have a form of cerebral syncope in which she had dysregulation of cerebral blood flow to upright posture, especially with orthostatic stress. Id. at 2.

On August 4, 2010, petitioner saw Dr. Frisca Yan-Go, a neurologist, at UCLA Health System on the recommendations of Dr. Cintron and Dr. Atiga. Med. recs. Ex. 18, at 2. Petitioner saw Dr. Yan-Go for dysautonomia, the main component of which was syncopal attack when standing, but also inability to eat or drink without her body expelling food or liquid. She needed to engage in 10 minutes or more of aerobic activity before she could tolerate eating.

[59] Low levels of epinephrine and norepinephrine can contribute to a variety of physical and mental conditions, including: anxiety, depression, fibromyalgia, hypoglycemia, migraine headaches, restless leg syndrome, and sleep disorders. Chronic stress, poor nutrition, and taking certain medications, such as methylphenidate (Ritalin) can make someone produce less epinephrine and norepinephrine. What's the Difference Between Epinephrine and Norepinephrine?, HEALTHLINE NEWSLETTER, https://www.healthline.com/health/epinephrine-vs-norepinephrine (last visited Jan. 8, 2019).

Once she stopped exercising, she could not eat and quickly lost weight and became severely dehydrated. She is very intolerant to heat and humidity. She cannot lift her legs to walk, her speech becomes slurred and she has nausea, dizziness, and profuse sweating. After eating, she gets very dizzy. Sometimes, when she has to talk or gets short of breath with chest pain, this triggers a dystonic reaction in which her jaw tightens, she slurs her words, and she adopts abnormal positioning of her extremities, all while she is conscious. She had a tilt-table test on July 15th and apparently her blood pressure did not change much, but her heart rate increased from 70 to 125, and she had presyncope, resulting in stopping the test. Id.

Dr. Yan-Go went through petitioner's history. She received her first flu shot in October 2007 and had flu symptoms. She received her second flu shot in October 2008 and developed a flu and bronchitis pneumonia 30 days after the shot. On September 3, 2009, she had her third seasonal flu shot [this is incorrect; her flu vaccination in 2009 occurred on August 23, 2009; Dr. Yan-Go is basing this history on petitioner's recounting her history to her]. Petitioner said she had total body aches and fatigue. Nine days later, on September 12, 2009, she could not move much, was very fatigued, and went to the hospital which said she had increased creatine kinase and diagnosed her with rhabdomyolysis. On September 17, 2009, she saw an infectious disease specialist who thought she might have Lyme disease. Her speech got worse and she felt fatigued and weak. In retrospect, they thought she might have a form of postinfectious GBS.

On September 27 and 28, 2019, petitioner had low orthostatic tolerance and orthostatic hypotension, but not a full faint. By October, she had to lie down most of the time. In December 2009, she fainted again. In March 2010, she saw an endocrinologist and other specialists who said she might have a form of GBS-like symptoms which triggered a dysautonomia. The dysautonomia fluctuated in intensity intermittently. She could not change position from sitting or bending to standing without being dizzy or fainting. Norpace controlled it 60 percent. She cannot hold her breath for more than five seconds without becoming dizzy and then she loses some speech and the ability to walk. Even at 75 degrees in temperature, she has extreme fatigue and then chest pain and headache especially when she was walking and standing. She slept quite well and sometimes had to sleep for 10 hours at night and four hours in the day. Id.

She gets depressed if she cannot exercise. She cannot tolerate stress without sharp chest pain, slurred speech, weakened muscles, and dizziness. She has extreme difficulty eating and consuming or retaining water if she cannot complete 10 to 20 minutes of exercise like running, biking, rowing, or stair stepping. Then her symptoms would return and she could not tolerate food. She cannot control her leg muscles without Neurontin. Sometimes, she can soothe the dystonia with a sensory trick like touching her left eye. She cannot tolerate multiple noises, sounds, or flashing lights, which result in a "violent convulsion," but she has 90 percent control with low-dose Neurontin. She cannot multitask and has intermittent difficulty speaking, which speech therapy did not improve. Id. Many times, she has extreme difficulty with cognition concerning calculating, remembering things, strategizing, and recalling, but she did not mention whether she improved in a supine position from being upright. Id. at 2-3. Her Epworth Sleepiness Scale was 15, with normal being less than 9. Id. at 3. She has been observed to have

sleep talking and sleep walking. Her Beck Inventory[60] is rated slightly elevated at 14 with normal being less than 9. Id.

On physical examination, Dr. Yan-Go notes that petitioner was not orthostatic. Id. at 3. Dr. Yan-Go hyperventilated her for only 10 seconds. Her eyes looked glassy, but she was still conscious and then she went into almost a tetany[61] state. Her jaw locked and her extremities went dystonic for about 10 to 15 seconds. Dr. Yan-Go told her to close her mouth and stop hyperventilating. Petitioner then came back to baseline in 30 seconds. Dr. Yan-Go wrote this was more of a tetany reaction to respiratory alkalosis and not the usual chronic dystonic syndrome because petitioner did not have dystonic posturing in baseline except when she was being hyperventilated. Dr. Yan-Go put petitioner on a bed and was able to get her to breathe very slowly. She was able to breathe about 20-30 seconds without dystonic posturing if she breathed slowly and was recumbent. Id. Dr. Yan-Go noted petitioner did not have Raynaud's phenomenon[62] and had some increased sweating while she was hyperventilating.

In her assessment, Dr. Yan-Go writes:

> This is a very complex set of symptomatology. It is very hard to put it all together, but the way I analyze it is that she is born with normal nervous system and autonomic nervous system.

Id.

On August 25, 2010, petitioner saw Dr. Yan-Go again. Id. at 1. Dr. Yan-Go states petitioner "has a very complex symptomatology, many of which I cannot explain by unified disorder." Id. Dr. Yan-Go thought petitioner's tilt-table test was interesting because when she was tilted, she had dystonic posturing and slurred speech. Dr. Yan-Go thought petitioner had some symptoms of orthostatic intolerance or maybe POTS, and might have some presyncopal phenomena, but not full syncopal phenomena. If petitioner did not exercise vigorously for 30 minutes, she could not get a sip of water or a bolus of food without having slurred speech and she would get very dystonic and fall. She had neurologic testing, imaging, and neuropsychologic testing without a diagnosis. She was referred to Dr. Yan-Go for dysautonomia. Dr. Yan-Go said she would not be able to explain all of petitioner's symptoms such as walking sideways or else she gets dystonia. Dr. Yan-Go observed all these events that day in her office. Petitioner took herself off Neurontin and Norpace in the prior two weeks because she had to do that before undergoing autonomic testing. She told Dr. Yan-Go that she could not tolerate the heat because she is intolerant of heat and cold temperatures and barometric pressure. Petitioner could not

[60] Beck Depression Inventory is "a self-report questionnaire for measuring the symptoms of depression, focusing on the cognitive symptoms." Dorland's at 955.

[61] Tetany is "hyperexcitability of nerves and muscles due to decrease in concentration of extracellular ionized calcium, which may be associated with such conditions as parathyroid hypofunction, vitamin D deficiency, and alkalosis or result from ingestion of alkaline salts; it is characterized by carpopedal spasm, muscular twitching and cramps, laryngospasm with inspiratory stridor, hyperreflexia, and choreiform movements." Dorland's at 1904.

[62] Raynaud's phenomenon is "intermittent bilateral ischemia of the fingers, toes, and sometimes ears and nose, with severe pallor and often paresthesias and pain, usually brought on by cold or emotional stimuli or anatomical abnormality." Dorland's at 1430.

swallow one gulp of water in front of Dr. Yan-Go without exercising for 30 minutes. Then she got dystonic speech. Dr. Yan-Go wrote, "I am really out of ideas today and so I said if she needs to resume her Neurontin and Norpace so that she could eat and exercise 30 minutes but not hyperventilate so she would not faint," she could do so. Dr. Yan-Go suggested petitioner see a gastroenterologist and take some swallowing tests and motility[63] testing to determine if she has dysmotility of the GI tract. Dr. Yan-Go said she "entertained" the idea that petitioner might have dysautonomia, but Dr. Yan-Go observed petitioner did not have seizures because petitioner could talk, this was gradual, and petitioner came back with no change of her sensorium. In addition, petitioner had an EEG during these spells and the EEGs were normal. "I am worried whether this also has some functional overlay and subconscious effect of conversion reaction or not." Id. Dr. Yan-Go stated it was very difficult, and whether petitioner had true triggers for movement disorder, dystonic movement was "very very hard to differentiate." Id.

On August 26, 2010, petitioner saw Dr. Kevin Ghassemi, a gastroenterologist, at UCLA. Id. at 8. Petitioner complained that she would vomit if she had not exercised for 30 minutes prior to eating. Id. However, if she exercised for 30 minutes, she was able to swallow solid food and liquid for the next 24 hours. Id. In addition, she felt a sense of throat constriction, dizziness, sweating, chest discomfort, and some slurring of her speech. Id. Petitioner reported that even drinking water could immediately lead to slurring of speech. Id. Dr. Ghassemi reviewed her laboratory testing. Petitioner had a normal C-peptide, morning cortisol level, metabolic panel, lipid panel, and renin-angiotensin-aldosterone[64] labs. Her ACTH level was slightly below the reference range. Id. On physical examination, when Dr. Ghassemi palpated petitioner's carotid arteries, she developed slurred speech which immediately improved on lying in the supine position. Id. at 9. She had a normal gait except for an episode of slurred speech when she appeared to have unsteadiness. When she developed episodes of slurred speech during carotid palpation and when she provoked symptoms by drinking water, she had twitching of her eyes and lips, was unable to stick out her tongue, and had unsteady gait. Id. Dr. Ghassemi's impression was, "Dysphagia[65] and vomiting in the setting of a complex group of symptoms." He found no reason to conclude petitioner had a structural gastrointestinal abnormality, given that she experienced periods of tolerating both solids and liquids without dysphagia. He stated petitioner might have a motility disorder but in the setting of her other symptoms, "it is unlikely that she has a primary gastrointestinal motility disorder." Id.

On August 30, 2010, petitioner returned to Dr. Ghassemi after undergoing esophageal manometry[66] and an upper GI series, both of which were normal. Id. at 11. Both tests provoked a feeling of dysphagia and nausea. Since her manometry, after each meal, within 10 to 15 minutes, petitioner began to experience diarrhea while she was lying down. Subsequently, while she was sitting to defecate, she would start to vomit. She recalled having similar problems

[63] Gastric motility is "the spontaneous movements of the stomach muscles that grind food and mix it with gastric secretions, and move the products into the duodenum." Dorland's at 1182.
[64] Renin-angiotensin-aldosterone system (RAAS) is "the regulation of sodium balance, fluid volume, and blood pressure by renal secretions." Dorland's at 1862.
[65] Dysphagia is "difficulty in swallowing." Dorland's at 579.
[66] Manometry is "the measurement of pressure by means of a manometer." Dorland's at 1104. A manometer is "an instrument for measuring the pressure or tension of liquids or gases." Id.

lasting about four days when she took an SSRI[67] for her symptoms. She worried about becoming dehydrated. On physical examination, petitioner went in and out of speaking with a British accent. Petitioner asked Dr. Ghassemi if she had postprandial[68] hypotension and he said he could not find in the literature a specific controlled way of making this diagnosis. Dr. Ghassemi assumed petitioner had an underlying neurologic disorder, "possibly related to autonomic dysfunction" without evidence of esophageal dysmotility. Id.

On September 9, 2010, petitioner had an evaluation of autonomic disorder at The Ohio State University Department of Neurology. Med. recs. Ex. 13, at 13 and Ex. 37, at 1-2. The tilt test findings were consistent with grade II orthostatic intolerance or POTS. Med. recs. Ex. 37, at 2. The technologist conducting the test commented that petitioner "experienced symptoms of feeling hot, focused in her upper trunk and head, with slight diaphoresis.[69] The patient's hands and feet were cold and moist to the touch." Id. at 1. Dr. Sheri Hart wrote that the tilt test findings were consistent with Grade II Orthostatic Intolerance (Postural Orthostatic Tachycardia Syndrome). Id. at 2. Petitioner's heart rate-deep breathing study was normal. Her blood pressure and pulse pressure were normal, but variable during 15 minutes of orthostatic stress. Her heart rate variability in response to deep breathing was normal. Id.

On September 14, 2010, petitioner saw Dr. Yan-Go. Med. recs. Ex. 18, at 6. Dr. Yan-Go reviewed Dr. Kevin Ghassemi's complete GI test. Petitioner did not have a structurally abnormal GI problem and no true dysfunctional GI problem in motility. Dr. Yan-Go's opinion was that petitioner did not have a serious, pure, autonomic failure or degenerative dysautonomia. Dr. Yan-Go was confident that she could treat petitioner's symptoms to prevent deconditioning and prevent chronic patterning in her brain that might lead to further disability. Dr. Yan-Go listed two choices. First, petitioner could have another full diagnostic study at the Mayo Clinic, but the clinic cannot manage her treatment. Second, she can manage each symptom locally. Dr. Yan-Go suggested nutritional management and directed petitioner to the website mypyramid.gov to look at a high-protein, complex-carbohydrate, nutrient-dense diet with fluids and small feedings. Dr. Yan-Go said petitioner had to condition her nervous system. She told petitioner not to do aerobic exercises. Dr. Yan-Go preferred petitioner do aquatic exercise in an indoor pool with controlled light, humidity, and temperature. Dr. Yan-Go said petitioner might have to repattern her brain by seeing a psychologist for repatterning or cognitive behavioral therapy. She also had to have proper sleep. Id.

On October 4, 2010, petitioner saw Dr. Robert A. Wohlman at NW Gastroenterology Associates with the chief complaint of postprandial hypotension. Med. recs. Ex. 42, at 16. Petitioner stated that "whenever she eats, within 15 minutes she becomes allergic, dizzy, diaphoretic, and sweaty." Id. Petitioner had to have her feet elevated after eating to help control her symptoms. He described petitioner's medical condition as "unusual." Id. Petitioner said she developed a "huge amount of neuropathy" after having a flu shot several years previously. Id. Petitioner did not have any anorexia, weight loss, fever, musculoskeletal complaints, or other

[67] SSRI stands for selective serotonin reuptake inhibitor. Dorland's at 1759.
[68] Postprandial means after a meal. Dorland's at 1502.
[69] Diaphoresis is sweating. Dorland's at 509.

gastrointestinal or systemic complaints. Id. However, under review of systems, Dr. Wohlman listed petitioner complained of diarrhea, abdominal pain, constipation, nausea, vomiting, and getting full quickly at meals. Id. at 17. Petitioner's gait was normal and she could undergo exercise testing and/or participate in an exercise program. Id. at 18. Petitioner's memory was intact for recent and remote events. Dr. Wohlman's assessment was that petitioner had autonomic dysfunction manifested especially postprandially. Id.

On October 28, 2010, petitioner saw Dr. Cintron, her neurologist. Med. recs. Ex. 1, at 2. Her voice and walking were better. Her problem continued to be memory and decompensating when she tried to work hard cognitively. In addition, she had fainting episodes when she ate. Id.

On December 15, 2010, petitioner saw Dr. Daniel V. Wilkinson Jr., a cardiologist at Swedish Medical Center. Med. recs. Ex. 12, at 3. Under history of present illness, he wrote petitioner had a very unfortunate history of a profound immunologic reaction to a flu shot, resulting in autonomic dysfunction which she characterized as POTS. She responded reasonably well to Norpace. She subsequently developed profound postprandial hypotension. She manifested virtual collapse with loss of speech and was placed on Sandostatin[70] intramuscularly, causing "a dramatic improvement." Id. Petitioner was extremely heat intolerant and reported that if she walked into a heated building, her blood pressure would drop and she would become presyncopal. She did not have a history of palpitations. Id.

On January 14, 2011, petitioner saw Dr. Cintron, who noted that petitioner was able to walk better, run better, and tolerate food significantly better. Med. recs. Ex. 1, at 1. Dr. Cintron stated the biggest cognitive problem petitioner still had was speaking in her accent which was more prominent, and cognitive function when she was in a hot temperature and standing, compared to when she was lying down. Id.

On February 3, 2011, Dr. Cintron wrote a medical summary. Med. recs. Ex. 26, at 1. He states, "In summary, her medical and neurological situation has been at best confusing and very difficult to intellectually define." Id. He concludes that her main physiological pathology involved her autonomic nervous system and regulation of cerebral blood flow, resulting in extreme sensitivity to collapse and fainting for which she tried to compensate by exercising "almost to a pathological amount" and taking medications to augment vasomotor tone. Id. Because of ongoing cerebral hypoperfusion, she developed cognitive processes and issues which fluctuated and changed her intonation. Dr. Cintron states, "I don't know if these particular problems are related to the initial insult, versus ongoing cerebral hypoperfusion. All of this is in my opinion is casually [sic] related to her flu vaccination, which is not unknown to cause post-

[70] Sandostatin is "trademark for preparations of octreotide acetate." Dorland's at 1667. Octreotide is "an eight-amino acid synthetic analogue of somatostatin, having actions similar to those of somatostatin but having a prolonged duration of effect." Id. at 1312. Octreotide acetate is "the acetate salt of octreotide, used as a treatment adjunct for the palliative treatment of diarrhea associated with gastrointestinal endocrine tumors." Id. Somatostatin is "any of several cyclic tetradecapeptides elaborated primarily by the median eminence of the hypothalamus and by the delta cells of the pancreatic islets; they inhibit release of growth hormone, thyrotropin, and corticotropin by the adenohypophysis, of insulin and glucagon by the pancreas, of gastrin by the gastric mucosa, of secretin by the intestinal mucosa, and of renin by the kidney." Id. at 1735.

vaccination neuropathies, which can present with autonomic instability." Id. He goes on to state that petitioner is not able to be gainfully employed and is totally disabled. Id.

On February 3, 2011, petitioner saw Dr. Wilkinson. Med. recs. Ex. 12, at 1. Petitioner continued to deal with abnormal neurovascular activity with symptomatic hypotension. She was taking very potent medications. She had multiple "very unusual symptoms" including changes in her speech and language patterns associated with presumed drop in blood pressure. Her exercise tolerance was poor. She stated walking up two flights of stairs caused her to lose her speech and become presyncopal. She started taking midodrine,[71] but it was difficult to know if she had a significant response. She also took disopyramide[72] and gabapentin. She took Sandostatin which apparently allowed her to eat reasonably. She denied palpitations. She did not have unusual syncope. She was frequently presyncopal presumably on an orthostatic basis. She said she had episodic chest pain which totally went away with exercise and she felt best when exercising vigorously. Id. Dr. Wilkinson's impression was that petitioner had severe vasodepressive syndrome, "apparently following viral syndrome," and "unusual manifestations" of hypotension which in addition to presyncope included change of voice and dialect. Id. at 2.

On February 15, 2011, petitioner wore a Holter monitor[73] for 23 hours and 55 minutes because of syncope and collapse. Id. at 5. Petitioner reported symptoms of shortness of breath and chest pain, but the monitor showed they were clearly not related to arrhythmia. Id.

On July 29, 2011, petitioner was evaluated by Dr. Blair Grubb at the Cardiac Electrophysiology and Autonomic Function Clinic at the University of Toledo Medical Center. Med. recs. Ex. 38, at 1. Dr. Grubb writes petitioner developed POTS because of flu vaccination. Dr. Grubb was under the impression that petitioner received flu vaccine on September 3, 2009, rather than the actual date which was August 23, 2009. Therefore, his statement that "shortly thereafter" (i.e., shortly after the vaccination), petitioner began to experience muscle weakness, fatigue, and exercise intolerance as well as POTS is inaccurate. Petitioner continued to misinform Dr. Grubb because he notes petitioner was diagnosed with GBS as a consequence of vaccination.[74] Id. On physical examination, petitioner's supine blood pressure was 148/78 and her upright blood pressure was 148/84. Her pulse in both supine and upright positions was 79. Id. To Dr. Grubb, "it sounds as though she has developed an autonomic neuropathy" as the result of a reaction to the vaccination. Id. at 1-2. Dr. Grubb stated that he had "personally seen this on several occasions previously" and "in most cases of postural tachycardia syndrome in adults, they were well either until a viral infection or similar immunologic stimulus caused them to produce autoantibodies against peripheral acetylcholine receptors in the sympathetic and parasympathetic system." Id. at 2. Dr. Grubb suggested adding pyridostigmine (Mestinon) to

[71] Midodrine hydrochloride is "a direct-acting sympathomimetic agent, which stimulates the α-adrenergic receptors of the arteriolar and venous vasculature; used as a vasopressor in the treatment of orthostatic hypotension." Dorland's at 1165. A vasopressor stimulates "contraction of the muscular tissue of the capillaries and arteries." Id. at 2027.

[72] Norpace. See supra, n.58.

[73] A Holter monitor is a type of ambulatory EKG. Dorland's at 1175.

[74] Petitioner's history to Dr. Grubb and his reliance on it are inconsistent with petitioner's medical records where her treating doctors ruled out GBS. Med. recs. Ex. 44, at 17.

petitioner's medical treatment of Neurontin, Norpace, midodrine, and octreotide. While noting Mestinon was initially used to treat myasthenia gravis, its use has extended to autonomic nervous system disease. For petitioner's cognitive impairment, he prescribed Cerefolin NAC. He described it as a modified form of folic acid frequently used to treat cognitive impairment associated with chemotherapy. Dr. Grubb spent two and one-half hours with petitioner. Id.

On August 12, 2011, petitioner had a single-photon emission computed tomography ("SPECT") brain scan at Cedars-Sinai Medical Center that Dr. Kelly E. Williams, petitioner's new PCP, requested. Med. recs. Ex. 39, at 2-3; Ex. 52, at 5-6; Ex. 54, at 13. According to the report, Dr. Alan D. Waxman noted marked reduction in perfusion of the watershed[75] areas of the frontal lobes extending to the posterior brain and a decrease in perfusion in the right thalamus when compared to the left. Med. recs. Ex. 39, at 2, and Ex. 54, at 13. The periventricular white matter/ventricular regions were within normal limits. There was a slight but definite decrease in the right cerebellum when compared to the left. Dr. Waxman's impression was that the findings were consistent with a vascular process, mainly impacting the small vessels and, to some extent, branches of the right middle cerebral artery and left posterior cerebellar artery. Dr. Waxman stated these findings were highly associated with systemic lupus or other autoimmune processes, including antiphospholipid antibody syndrome. Id. Lyme disease was a consideration as well. Med. recs. Ex. 39, at 3.

However, petitioner's laboratory testing for antiphospholipid antibodies, C-reactive protein, Cyclic Citrullinated Peptide ("CCP") antibody, and ANA choice Cascade antibody were all normal. Med. recs. Ex. 41, at 1-5, and Ex. 42, at 30-37. A handwritten note on the laboratory test report stated ". . . blood tests all normal. No evidence of autoimmune disease." Med. recs. Ex. 42, at 30. The filing of Exhibit 42 lists more blood tests than the filing of Exhibit 41 although they both reflect testing done on August 31, 2011 with reports issued on September 6, 2011. Exhibit 42 includes petitioner's negative testing for not only phosphatidylserine antibodies, but also cardiolipin antibodies, IgG, IgM, and IgA. Med. recs. Ex. 42, at 31.

On November 2, 2011, petitioner saw Dr. Elmer Y. Chang, a gastroenterologist, in Mission Viejo, California, upon Dr. Williams' referral for an evaluation for postprandial hypotension. Med. recs. Ex. 114, at 1. In August 2010, petitioner went to UCLA for an upper endoscopy, CT scan of her abdomen and pelvis, esophageal manometry, and a gastric emptying study, all of which were reportedly normal. Id. at 2. She was diagnosed with postprandial hypotension secondary to autonomic dysfunction. Id. She was started on octreotide with dramatic improvement of her symptoms. Id. at 2-3. Since then, she had not had any further GI symptoms. Id. at 3. Dr. Chang found petitioner's vitals and physical examination unremarkable. His impression was:

> This is a very unusual case of postprandial hypotension. Normally, one would expect [a] slight decrease in systemic blood pressure following meals due to increased splanchnic blood flow.

[75] Watershed is "an area where the peripheries of two vascular beds meet, particularly in the brain." Dorland's at 2076.

> However, because of her autonomic dysreflexia, she develops an exaggerated blood pressure response. The reason why octreotide may work for her is that it decreases splanchnic blood flow, thereby increasing systemic blood pressure. Obviously, large meals would also increase splanchnic blood flow and should be avoided.

Id. Dr. Chang recommended petitioner continue with octreotide, which his clinic would administer to her. In addition, she should eat small meals multiple times a day instead of large meals so as to decrease the chance of postprandial hypotension. Id.

On December 8, 2011, petitioner saw Dr. Mariko L. Ishimori, a rheumatologist, at Cedars-Sinai Medical Center, for an assessment of her abnormal SPECT scan and concern for a possible central nervous system vasculitis. Med. recs. Ex. 54, at 4. Petitioner told Dr. Ishimori that she had had a severe and unusual reaction to flu vaccination resulting in an autoimmune reaction two years previously. It started with postprandial hypotension and progressed to orthostatic hypotension which required hospitalization. Within the first few days of hospitalization, she developed rhabdomyolysis with CPK peaking at 13,000. She said all these symptoms progressed and accelerated over one week, escalating over a two-month period during which she was hospitalized consistently at Inova Fairfax Hospital. Id.

Serologic testing resulted in a negative result for an extensive panel of antiphospholipid antibodies. Petitioner's ANA was negative as well. Petitioner stated that she had only one positive ANA test during her acute hospitalization, and the highest was 1:80. Id. Dr. Ishimori ordered ANA testing on December 8, 2011. Med. recs. Ex. 57, at 1. Two results are listed: 1:320 (speckled pattern) and 1:40 (homogeneous pattern). Petitioner tested negative for the following: anti-centromere antibody, anti-SCL-70 antibody, anti-Sm antibody, anti-RNP antibody, anti-RO antibody, anti-LA antibody, anti-DS DNA antibody, and anti-chromatin antibody. Anti-DS DNA antibody tested 3.0 when normal is less than 7.0. Petitioner's complement C3 tested at 110 when normal is between 79 and 152 mg/dL. Petitioner's complement C4 tested at 25 when normal is between 16 and 49 mg/dL. Petitioner tested negative to cardiolipin, IgG, IgA, and IgM. She tested negative to citrulline, rheumatoid factor, and thyroid microsomal P antibody. Id.

Petitioner said she tried to exercise 20 hours per week by running, cycling, and weightlifting. Id. at 5. She did not have a problem with sleep. Petitioner said she had an allergy to lidocaine,[76] Carbocaine,[77] Ativan, Zoloft,[78] Klonopin, transdermal Scopolamine patch, Benadryl,[79] and NyQuil, all of which resulted in heart palpitations, slurred speech, fainting, and

[76] Lidocaine is "a drug having anesthetic, sedative, analgesic, anticonvulsant, and cardiac depressant activities, used as a local anesthetic." Dorland's at 1034.

[77] Carbocaine is "trademark for preparations of mepivacaine hydrochloride." Dorland's at 288. Mepivacaine hydrochloride is "a local anesthetic, an analogue of lidocaine." Id. at 1136.

[78] Zoloft is "trademark for preparations of sertraline hydrochloride." Dorland's at 2092. Sertraline hydrochloride is "a selective serotonin reuptake inhibitor, used to treat depressive, obsessive-compulsive, and panic disorders." Id. at 1699.

[79] Benadryl is "trademark for preparations of diphenhydramine hydrochloride." Dorland's at 208.

difficulty breathing. Id. Dr. Ishimori did a review of systems. Petitioner had fatigue, weakness, pain, ringing in her ears, and dry mouth. Id. She claimed irregular heart beat and chest pains. Id. at 6. She had occasional shortness of breath and difficulty breathing at night. She had nausea and recurrent diarrhea. She had morning sickness lasting one hour, and joint pain involving her hands and feet. She had headaches, dizziness, fainting, muscle spasm, loss of consciousness, memory loss, and night sweats, all related to orthostatic hypotension. She had excessive thirst. Id.

On physical examination, petitioner had a blood pressure of 122/79. She was well-nourished. She did not have synovitis in the MCP,[80] PIP,[81] or DIP[82] joints. Her hands had full range of motion. She did not have evidence of tenderness over the extensor tendons and no evidence of tenosynovitis or erythema on the dorsum of her hands. She did not have warmth, swelling, or tenderness to palpation. She had full range of motion. Her ulnar nerve when examined led to significant tremor. She had no evidence of vascular changes. Her strength was intact bilaterally and symmetrically in the upper and lower extremities. She did not have evidence of muscle weakness. She had good strength, 5/5 symmetrically. Id.

Petitioner showed Dr. Ishimori the results of her lab tests done August 31, 2011. Id. at 7. Her antiphospholipid antibody panel was negative, as were antibody IgG, IgA, C-reactive protein, CCP antibody, ANA, and a complete metabolic panel. Dr. Ishimori discussed with petitioner that there was no clear-cut evidence based on her examination or history that she actually had lupus or a phospholipid antibody syndrome. Id. As far as her abnormal brain SPECT was concerned, Dr. Ishimori wrote "it is interesting to note that she had a normal MRI and MRA." Id. Although the SPECT scan was reported to be associated with some cognitive function and central nervous system lupus, Dr. Ishimori believed there were other potential etiologies and explanations, particularly in a patient with a very unusual history of a very severe orthostatic dysfunction, which may lead to poor episodic perfusion of specific areas of the brain, which might be related to her poor previous control of orthostatic hypotension. Id.

Dr. Ishimori recommended petitioner see Dr. Patrick D. Lyden, the chairman of neurology at Cedars-Sinai Medical Center. Id. Dr. Ishimori wrote:

> I think based on her examination I see no evidence of a peripheral autoimmune or connective tissue disorder that would explain a SPECT that would be vasculitic in nature, and I do not think that this result necessarily correlates with a vasculitis in this patient. In addition, I do not think this is a primary central nervous system vasculitic process as she is not in a typical group, and given her history of orthostatic hypotension . . . . I also discussed with the patient that in young women, … there are other conditions such as

Diphenhydramine is "a potent antihistamine (H_1 receptor antagonist) with anticholinergic, antitussive, antiemetic, antivertigo, antidyskinetic, and sedative actions." Id. at 523.

[80] MCP stands for "metacarpophalangeal." Med. Abbrev. at 221.

[81] PIP stands for "proximal interphalangeal." Med. Abbrev. at 282.

[82] DIP stands for "distal interphalangeal." Med. Abbrev. at 115.

> benign angiopathy that can occur, which are associated with more vasoreactivity than necessarily vascular inflammation, which can be mimics for vasculitic pictures. I think in her case most likely these abnormalities represent her perfusion related issues and not a true inflammatory vasculitic process.

Id.

On January 10, 2012, petitioner saw Dr. Patrick D. Lyden, chairman of neurology at Cedars-Sinai Medical Center. Med. recs. Ex. 54, at 9. Dr. Lyden did a physical examination and wrote petitioner was alert and fluent and had good recall. On looking to the right, petitioner had a few beats of rotary nystagmus, fast component. Id. On motor examination, she had normal bulk, power, and tone throughout. Id. at 10. On sensory examination, she was intact to light touch, pinprick, double simultaneous stimulation, and position sense. Her reflexes were markedly suppressed throughout with slight asymmetry, being more depressed on the right than on the left. Her finger-nose-finger coordination was normal. She had some functional component and stimulated tremor, but no deficit. Heel-shin was normal. Romberg[83] was negative. Heel-toe walking was normal. Id.

Dr. Lyden's impression was that:

> This is a very complicated case. I believe we need to assemble more data to be sure. Taking everything at face value, it sounds as though she did have an illness that has left her with a severe dysautonomia. In addition, I believe she has migraine as evidenced by the intolerance of bright lights, loud sounds and a history of migraines. A lot of her headache and nausea in response to bright lights could be migraine related. The dysautonomia has been documented and could be an exaggeration of normal vagal hypersensitivity. In addition, I believe there is an unsuspected psychiatric diagnosis, but it is too soon to be sure that this is real. She does have some trace neurologic findings that would localize to the right cerebellum or brainstem, given the rotary nystagmus and hypotonia. I would like to review her MRI scan.
>
> She will return next visit with MRI scans from Johns Hopkins and UCLA. We will perform a transcranial duplex to try to recreate the small-vessel disease seen on SPECT scan. I will review her medical workup at that time.

Id. at 10.

On January 13, 2012, petitioner returned to Dr. Lyden and had a transcranial doppler to see if she had small vessel vasculitis. Id. at 11, 12. Velocities and resistances were obtained at

[83] The Romberg sign is "swaying of the body or falling when standing with the feet close together and the eyes closed; the result of loss of joint position sense." Dorland's at 1715.

multiple depths from the middle cerebral, anterior cerebral, and posterior cerebral arteries. Id. at 11. In addition, the ophthalmic, vertebral, and basilar arteries, and extracranial internal carotid arteries were imaged. All of petitioner's waveforms were normal. Her velocities were slightly elevated, especially at depth. For example, at 51mm in the right middle cerebral artery, the pulsatility index was 1.34 and, on the left, 1.13. Dr. Lyden's impression was that petitioner's study was normal. However, the pulsatility indices trended toward the high side, indicating possible small vessel involvement. Id. His clinical impression was that, taken in the context of petitioner's other findings, the result of the transcranial doppler could indicate a normal state. However, petitioner could also have distal compression of the smallest vessels in her brain. Id.

On January 13, 2012, petitioner resumed her visit with Dr. Lyden. Id. at 12. Petitioner told Dr. Lyden she did not have any change in her symptomatology. She brought a series of medical records which Dr. Lyden reviewed. Petitioner's tilt test was negative for hypotension. During the tilt test, she had acute onset of dysarthria and dystonic posturing. Her multiple MRI scans were normal. Cardiologists treated her possible dysautonomia with Norpace. The medical records did not support any evidence of GBS. The medical records did not support any evidence of hypotension. Dr. Lyden notes the medical records repeatedly described petitioner's "bizarre and unusual symptoms related to postural changes." Id. He noted petitioner had a significant dystonic and syncopal reaction during a carotid ultrasound. Id.

On physical examination, petitioner's mental status was "again remarkable" for a significant indifference to her medical state. Id. She spoke in a foreign accent for approximately half of the visit. This resolved spontaneously and she finished the visit with her Midwestern American accent. Id. There was no change in her cranial nerve exam, her motor or sensory function, or her reflexes. Dr. Lyden's impression was that her transcranial doppler study was essentially normal but did have resistance indices trending toward the high side. After reviewing petitioner's medical records and her examination, Dr. Lyden stated he clearly saw that she had a number of symptoms that were "embellished;" however, she did have an acute reaction characterized by rhabdomyolysis during a dehydrational episode. Id. She also had symptoms that were suggestive of dysautonomia despite the negative tilt table test. Dr. Lyden believed petitioner needed a good psychologic evaluation, referring her to Dr. Ellen Basian. Id.

Dr. Lyden noted petitioner had a history of empty sella[84] on imaging, which he thought was probably benign, but recommended pituitary function testing. Dr. Lyden said there was an extremely remote possibility that petitioner could have acute intermittent porphyria.[85] That would explain the bulk of her symptoms, including the rhabdomyolysis. Petitioner was not at the time symptomatic, but nevertheless, he would send her for a baseline urine porphyrin and porphobilinogen[86] and see her in follow up after she saw Dr. Basian, the psychologist. Id.

[84] Sella is "a saddle-shaped depression." Dorland's at 1689.
[85] Porphyria is "any of a group of disturbances of porphyrin metabolism, characterized biochemically by marked increase in formation and excretion of porphyrins or their precursors and clinically by various neurologic and cutaneous manifestations." Dorland's at 1497.
[86] Porphobilinogen is "the immediate precursor of the porphyrins." Dorland's at 1497.

On February 29, 2012, petitioner saw Dr. Michael J. Olek, osteopath, at Newport Doctors Multiple Sclerosis Clinic. Med. recs. Ex. 61, at 1. Petitioner gave a history that the onset of her symptoms was August 23, 2009, the day of her flu vaccination. Id. Dr. Olek did not have petitioner's medical records. Petitioner complained that in January 2012, her extreme fatigue worsened. Id. Petitioner complained of blurry vision, eye pain, snoring, ringing in the ears, earache, chest pain, fainting, shortness of breath, nausea, vomiting, frequent urination, nighttime urination, sleepiness, headache, dizziness, numbness, fatigue, hot and cold intolerance, and joint pain. Id. at 3. Although petitioner complained to Dr. Olek that she was allergic to Lidocaine, Carbocaine, Zoloft, Ativan, Klonopin, transdermal Scopolamine patch, Benadryl, and Nyquil, he thought that most of these were just bad reactions, rather than allergies. Id. Petitioner was divorced and lived alone. She drank seven cups of coffee a day. Id.

On physical examination, petitioner had occasional slurred speech and foreign accent. Id. at 4. Her motor strength was 5 out of 5 with normal tone and bulk. Her deep tendon reflexes were +2 and equal. Her toes were downgoing. Coordination showed normal finger-to-nose-finger, rapid alternating movement, and heel-to-shin. Sensory exam was intact to light touch, pinprick, vibration, proprioception, and temperature. She was able to walk 25 feet in six seconds with a normal gait. Her tandem walk was normal. The Romberg was negative. Petitioner could hop independently on each foot. Dr. Olek's wrote his impression, "At this time, it is difficult to have a unifying diagnosis." Id. He felt petitioner had some symptoms of lupus and autoimmune dysautonomia. He planned to have her undergo a spinal tap to look for signs of MS. In December 2011, she had an ANA positive at 1:320 in a speckled pattern. Id.

On March 13, 2012, petitioner saw Dr. Chang for a follow-up visit. Med. recs. Ex. 114, at 4. Petitioner reported that she had more trouble with bloating with wheat products, but the trouble stopped when she stopped eating wheat products. Id. Dr. Chang ordered labs as well as recommended a trial of probiotics. Id. Noted on petitioner's pathology report, collected on March 20, 2012, was the recommendation that petitioner should proceed with a gastric emptying study. Id. at 6. A pathology report dated March 20, 2012 showed mild inflammation of petitioner's esophagus consistent with acid reflux. Id.

On March 28, 2012, petitioner saw Dr. Olek, complaining of left foot drop. Med. recs. Ex. 61, at 5. Petitioner had an MRI of her brain on March 15, 2012 whose result was normal. Id. at 5. Petitioner told Dr. Olek that two years previously, she underwent hyperbaric oxygen for one to two hours a day "prescribed by her neurologist" [Dr. Buttar is an osteopath, not a neurologist]. Id. She said it was helpful, but she stopped in December and related that, since January, she had been having increasing problems. Petitioner had a lumbar puncture. Her IgG index was normal at 0.52, and she did not have oligoclonal banding. Id.

Petitioner saw Dr. Swaraj Bose,[87] a neuro-ophthalmologist at University of California, Irvine, on February 22, 2012 and March 21, 2012. Dr. Olek had Dr. Bose's records. Dr. Bose's

[87] Petitioner did not file Dr. Bose's records. At the time petitioner saw him, Dr. Bose was Associate Professor of Ophthalmology and Neurology, a board-certified ophthalmologist, and Director of Neuro-ophthalmology and orbital surgery at the University of California, Irvine. He specializes in the medical and surgical management of patients with diseases of the eye in relation to the brain. His interests include optic neuropathies including MS, cranial nerve

records showed petitioner's visual acuity was normal at 20/20, and her color vision was normal at 8 out of 8 bilaterally. Her confrontational fields were normal. Id. Petitioner told Dr. Olek that Dr. Bose diagnosed her with optic neuritis, but Dr. Olek did not find that diagnosis in Dr. Bose's medical records. Id. at 5-6. In petitioner's visit to Dr. Bose on March 21, 2012, Dr. Bose wrote petitioner's eye exam was totally normal. Id. at 6.

During a physical and neurologic examination, Dr. Olek observed that petitioner was awake, alert, and oriented with fluent speech. Her visual fields were full. Motor strength was 5 out of 5 throughout except for her left lower extremity which was 5- out of 5 distally. Her deep tendon reflexes were +2 and equal. Her coordination was equal except for her left lower extremity. Her sensory exam was intact throughout. She was able to walk 25 feet in eight seconds, with a mild left foot drop. Id. Dr. Olek again noted it was difficult to have a unifying diagnosis; however, he felt petitioner had some symptoms of lupus and autoimmune dysautonomia. Id. He prescribed a trial of five days of one gram of IV methylprednisolone and continuation of her medications. He told her to continue with physical therapy. Petitioner said she was going to buy a hyperbaric oxygen chamber for $7,500 and would start this after her IV infusion at Hoag Hospital. Id.

On March 30, 2012, petitioner had a gastric emptying study for abdominal pain. Med. recs. Ex. 62, at 1. Dr. Sam Kipper wrote petitioner had significant retention of activity within the gastric fundus and remainder of the stomach. This was very slow gastric emptying. Petitioner's half-emptying time was 223 minutes compared to the normal time of between 30 and 90 minutes. Petitioner emptied 18 percent of the gastric mean by 90 minutes. Dr. Kipper's impression was markedly prolonged gastric emptying consistent with gastroparesis.[88] Id.

On April 13, 2012, petitioner had a CT scan of the abdomen and pelvis with and without contrast. Med. recs. Ex. 114, at 8. The test found the lung bases, liver, spleen, gallbladder, adrenal glands, pancreas, kidneys, ureters, bladder, uterus, adnexa,[89] abdomen, and pelvis were normal. Id. The test showed that there was mild distention of the gallbladder. Id. Petitioner then returned to see Dr. Chang on April 16, 2012. Id. at 10-11. Dr. Chang recommended petitioner continue her medications, eat small meals, and return in three months.[90] Id. at 11.

On April 16, 2012, petitioner saw Dr. Chang. Med. recs. Ex. 114, at 10. Her postprandial nausea and bloating improved with octreotide. Reglan[91] also helped. She had epigastric pain whenever she overate. Id.

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palsies, ocular myopathies including myasthenia gravis, thyroid eye disease, pseudotumor cerebri, and eyelid/orbital tumors. Swaraj Bose, UCI FACULTY PROFILE SYSTEM, https://www.faculty.uci.edu/profile.cfm?faculty_id=4861 (last visited Mar. 6, 2019).

[88] Gastroparesis is "paralysis of the stomach, usually from damage to its nerve supply, so that food empties out much more slowly, if at all. Symptoms include postprandial bloating and vomiting, and often hypoglycemia because of food not moving properly into the duodenum." Dorland's at 765.

[89] Adnexa are appendages. Dorland's at 32.

[90] Petitioner subsequently visited Dr. Chang for follow-up appointments to manage her gastroparesis and postprandial hypotension on May 29, 2012, April 9, 2013, and August 28, 2013. Med. recs. Ex. 114, at 12-16.

[91] Reglan is "trademark for preparations of metoclopramide hydrochloride." Dorland's at 1621. Metoclopramide hydrochloride is "a prokinetic dopamine receptor antagonist that stimulates gastric motility, used as an antiemetic,

On April 18, 2012, petitioner saw Dr. Daniel J. Wallace, an internist and rheumatologist in West Hollywood, CA, to rule out autoimmune disease. Med. recs. Ex. 60, at 8, and Ex. 112, at 1. He noted petitioner had an extremely complicated and detailed medical history. Id. Porphyria workup was negative. Id. Petitioner told him that a neuro-ophthalmologist told her she has optic neuritis but the doctor's notes do not mention it. On physical examination, she had a scanning speech pattern that changed into different foreign accents as the exam progressed. She was cushingoid.[92] Her carotid arteries were exquisitely sensitive to touch. Minimal Raynaud's was present. Her neurologic exam was intact. Id. Dr. Wallace's impression was that petitioner suffered from post-vaccination syndrome characterized by rhabdomyolysis and orthostatic hypotension in somebody with a strong family history for rheumatic disease. Med. recs. Ex. 60 at 9; Ex. 112, at 2. Dr. Wallace stated that petitioner may have a metabolic or mitochondrial myopathy that needed to be further explored. Id. He wrote, "I personally think she has 'Raynaud's of the brain'[93] with severe dysautonomia." Id.

… and in the treatment of gastroparesis and gastroesophageal reflux." Id. at 1154.

[92] Cushingoid means "resembling the features, symptoms, and signs associated with Cushing syndrome." Dorland's at 450. Cushing syndrome is "a complex of symptoms caused by hyperadrenocorticism due either to a neoplasm of the adrenal cortex or adenohypophysis, or to excessive intake of glucocorticoids. Symptoms may include adiposity of the face, neck, and trunk; kyphosis from osteoporosis of the spine; hypertension; diabetes mellitus; amenorrhea and hypertrichosis in females; … dusky complexion with purple striae; polycythemia; and muscular wasting and weakness. When secondary to excessive pituitary secretion of corticotropin, it is known as *Cushing disease*." Id. at 1827.

[93] Dr. Wallace wrote a letter in response to an article by Efrosini Papadaki et al., Neuropsychiatric Lupus or not? Cerebral Hypoperfusion by Perfusion-Weighted MRI in Normal-Appearing White Matter in Primary Neuropsychiatric Lupus Erythematosus, 77 ANN RHEUM DIS 441 (2018). David J. Wallace, Correspondence, 78 ANN RHEUM DIS e5 (2019). Dr. Wallace writes he read with interest the Papadaki article but believes the authors come to the wrong conclusion, firstly because they rely on definitions Dr. Wallace and others used in a 1999 article to define neuropsychiatric lupus syndromes, which Dr. Wallace now says are outdated and not evidence based. He cites to ACR Ad Hoc Committee on Neuropsychiatric Lupus Nomenclature, The American College of Rheumatology Nomenclature and Case Definitions for Neuropsychiatric Lupus Syndromes, 42 ARTHRITIS RHEUM 599 (1999). Dr. Wallace states in his correspondence related to the Papadaki article that the best objective measure for a central nervous system inflammatory process is a lumbar puncture. Dr. Wallace states that pleocytosis, increased protein levels, increased IgG synthesis rates, oligoclonal bands or antineuronal antibodies are the only objective metrics that can make the diagnosis of neuropsychiatric lupus outside a brain biopsy or obvious neuroimaging abnormalities. But none of the patients in Papadaki's articles had a spinal tap. Secondly, he criticizes Papadaki for not realizing that others before him, including Dr. Wallace's group, demonstrated that most lupus patients with neuropsychiatric lupus had SPECT imaging abnormalities consistent with hypoperfusion in the watershed regions. He cites to an article in which he is a co-author: Catherine B. Driver et al., Clinical Validation of the Watershed Sign as a Marker for Neuropsychiatric Systemic Lupus Erythematosus, 59 ARTHRITIS RHEUM 332 (2008). Dr. Wallace states in his correspondence, "In other words, most patients had vasomotor instability on an autonomic basis ('Raynaud's of the brain') in, for example, the frontal-parietal interface where the vasculature is very small, numerous and prone to spasm. While some of these patients have an inflammatory process, the majority develop 'lupus fog' as a consequence of intermittent hypoperfusion. This should not be considered to be neuropsychiatric lupus and is managed with cognitive behavioural [sic] therapy, anxiety reduction measures, biofeedback and approaches that target the dysautonomia of lupus." Dr. Wallace cites to Ljudmila Stojanovich, Autonomic Dysfunction in Autoimmune Rheumatic Disease, 8 AUTOIMMUN REV 569 (2009). Dr. Wallace also used the expression "Raynaud's of the brain" in an interview for a blog that one of his colleagues calls Attune Health. Dr. Wallace used the expression in relation to lupus patients complaining of a mental fog or difficulty thinking clearly. He explains that the sympathetic nervous system (part of the autonomic nervous system) controls opening and closing blood vessels. When it is cold, the patient's Raynaud's constricts the blood vessels. When it is warm, the blood vessels can over dilate and turn red. Dr. Wallace said the same phenomenon can occur in the brain. When

On May 17, 2012, Dr. Williams referred petitioner to Dr. Joey R. Gee, an osteopath at Mission Internal Medical Group in Mission Viejo, California, for consultation. Med. recs. Ex. 115, at 1. Petitioner complained about daily headache and sharp pains in her right eye. Id. Petitioner told Dr. Gee that she had optic neuritis in both eyes (there has never been a diagnosis of optic neuritis in petitioner; Dr. Gee did not have her neuro-ophthalmologist records to know that she did not have optic neuritis). Pyridostigmine was prescribed for possible myasthenia gravis but it did not help. Yet petitioner seemed to control her breathing problem, and weakness in her foot and voice. She reported a brain SPECT suggested vascular changes indicating small vessel vasculopathy, with impairment of the cerebellum, right thalamus, and bilateral watershed zones. Since January, petitioner had been declining, but her speech and drop foot were better. Id.

On physical examination, petitioner had normal range of motion in all four extremities. Her memory was intact. She had normal 5 out of 5 strength in her upper and lower extremities although she might have some fatiguing of the proximal muscles of the arms and hips. Id. at 4. Her reflexes were 2+ with absent frontal release signs. Id. Petitioner had normal tone, no spasticity, and normal sensation in the extremities. Id. at 5. Her balance was stable with her feet together and her eyes closed. Her gait was intact with tandem. Her balance was normal. She did not have any language deficits and had a normal attention span and concentration. Id.

Dr. Gee noted that petitioner presented with a myriad of symptoms, the underlying diagnosis was still an "enigma," and she had yet to present with a distinct primary diagnosis. Id. Petitioner had gross syndromes for the most part, "characterized by all the systemic complaints, ranging through a number of systems" of her body, i.e., rheumatological, cardiovascular, neurological, cognitive, orthopedic, ophthalmological, and gastrointestinal "to name a few." Id. Concerning the diagnosis of myasthenia gravis, Dr. Gee said it was difficult to include all her system complaints in that diagnosis. Dr. Gee thought lupus might encompass all these systemic complaints, but a rheumatologist did not give her a specific diagnosis. Id.

On May 29, 2012, petitioner saw Dr. Chang, the gastroenterologist. Med. recs. Ex. 114, at 12. She tried Domperidone,[94] which caused weakness and fatigue. Reglan seemed to help. Petitioner said when she runs, her gastroparesis improves. Petitioner was on Sandostatin LAR for postprandial hypotension. Dr. Chang put a question mark in his records as to whether petitioner's postprandial hypotension was secondary to autonomic dysfunction on his March 13, 2012 and May 29, 2012 records. Med. recs Ex. 114, at 4, 12.

On June 1, 2012, petitioner saw Dr. Gee, complaining of numbness and weakness. Med. recs. Ex. 115, at 8, 10. Petitioner underwent electromyography ("EMG") and nerve conduction studies ("NCS"). The impression was there was no electrodiagnostic evidence for diagnosing a

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the vessels overdilate, they cause a headache. When they overconstrict, they cause a mental fog. Dr. Wallace remarks, "I like to call it 'Raynaud's of the brain.'" Rheumatology. Getting Lost in the Brain Fog, THE PURSUIT OF BETTER, THE BLOG OF ATTUNE HEALTH, https://attunehealth.com/getting-lost-brain-fog/ (last visited Feb. 1, 2019).

[94] Domperiodone is "an antiemetic and prokinetic agent; its actions are related to its peripheral dopamine receptor-blocking properties. It is used in the treatment of upper gastrointestinal motility disorders caused by chronic and subacute gastritis and diabetes and the prophylaxis of gastrointestinal symptoms caused by dopamine agonists used to treat parkinsonism." Dorland's at 562.

neuromuscular process. Id. at 10. Repetitive stimulation of one isolated nerve did not reflect a decremental response which one would expect if petitioner had myasthenia gravis. Petitioner had stopped taking pyridostigmine for the testing. Id.

On July 18, 2012, petitioner visited Dr. Gee, complaining of gastroparesis and heart rate issues. She still complained of myasthenia gravis. Id. at 13. Dr. Gee told petitioner, "I counseled it is less clear as to the exact nature of her illness given neurological tests are unremarkable." Id. Petitioner appeared well on physical examination with no dizziness, no focal weakness, no gait disturbance, no memory impairment, and no paresthesia. Id. at 15. She did not have any language deficits. Id. at 16. Her knowledge was intact and she had normal attention span and concentration. Her cerebral angiogram was normal. Id.

On March 1, 2013, Dr. Williams filled out an evaluation report for Special Services, Saddleback College, to enable petitioner to receive disability-related support services. Med. recs. Ex. 174, at 1. Dr. Williams diagnosed petitioner with autonomic dysfunction. Id. at 2.

On March 8, 2013, petitioner reported to Dr. Gee, complaining of a cold over the prior two weeks and feeling weaker with trouble walking. Med. recs. Ex. 115, at 18. On physical examination, Dr. Gee found that petitioner did not have motor or sensory deficits. Her balance and gait were intact. Her coordination was intact and her reflexes were preserved at 2+. However, she had fatiguing weakness of her upper extremities and neck muscles, which caused spasms of her neck. Id. She did not have language deficits. Id. at 19. She had normal attention span and concentration. Dr. Gee thought her cold might have mildly exacerbated petitioner's myasthenia gravis. He prescribed a small dose of corticosteroid. To help her weakness, she was to use a smaller dose of pyridostigmine. Id.

On March 20, 2013, petitioner had a test for acetylcholine receptor binding antibody, which was negative (<0.30nmol/L), and a test for acetylcholine receptor blocking antibody, which was negative (<15). Id. at 22, 23. Also on March 20, 2013, petitioner had a test for MuSK on antibody titer which was negative. Id. at 24. The interpretation states, "This individual is negative for muscle-specific receptor tyrosine kinase (MuSK) antibodies that are associated with Myasthenia gravis syndrome (MG)." Id.

On March 22, 2013, petitioner was retested for acetylcholine receptor binding antibody, acetylcholine receptor blocking antibody, and MuSK on antibody, all of which were again negative. Id. at 26, 27, 29. In addition, on that date, petitioner was tested for acetylcholine receptor modulating antibody, which was negative at 9% (reference range: <32%). Id. at 28.

On March 26, 2013, petitioner signed a Saddleback College Special Services Educational Accommodations form, which Connie Jackson, M.S., signed as a faculty member of Special Services. Med. recs. Ex. 174, at 3. Ms. Jackson noted that petitioner needed to use a laptop for note-taking and written assignments because handwriting would be too difficult for her due to mobility issues. Id. Ms. Jackson also authorized petitioner to have use of a room with minimal distractions and an extension of time to include an additional half-hour increase to accomplish tasks. Id. at 4.

On April 9, 2013, petitioner saw Dr. Chang. Med. recs. Ex. 114, at 14. Petitioner said she had daily heartburn in the morning. She had postprandial nausea after breakfast. She was satiated until dinner time. She also vomited and had anorexia with a 10-pound weight loss since she last visited. Domperidone did not help her and she had not taken Reglan. Id. Dr. Chang started petitioner on Protonix[95] and restarted her on Reglan. Id.

On August 13, 2013, petitioner underwent a barium swallow and RAD UGI barium upper GI exams. Med. recs. Ex. 91, at 1. Dr. Jackson W. Penry wrote petitioner had a normal swallowing mechanism and no evidence for esophageal obstruction, ulceration, or mucosal mass lesion. She did not have hiatal hernia or gastric outlet obstruction. She had intermittent partial obstruction to the forward flow of contrast at the third portion of the duodenum. She did not have associated mucosal irregularity, external mass lesion, or fold thickening. The findings of intermittent partial obstruction might represent functional sequelae of external superior mesenteric artery. Id. Otherwise, this was a normal examination. Id.

On November 4, 2013, petitioner went to Mission Hospital ED in California with complaints of shortness of breath and weakness. Med. recs. Ex. 88, at 1. Petitioner told Dr. Raj Patel that she had a history of myasthenia gravis and autonomic dysfunction consisting of POTS, gastroparesis, intestinal pseudo-obstruction, and Raynaud's phenomenon. Her shortness of breath began on November 2, 2013 at 1:00 a.m. She said she had been sick for two weeks. But on Friday, her cat and dog fought and were sent to an animal hospital, triggering a flare of her myasthenia gravis. She woke up Saturday night at 1:00 a.m. with trouble breathing and called her neurologist Dr. Gee who advised her to come to the ED. Petitioner denied any pain, nausea, vomiting, chills, fever or dizziness. Id. at 1-2.

Later the same day, petitioner saw Dr. Loan T. Nguyen. Id. at 4. She told Dr. Nguyen that she had shortness of breath for the past three days. Dr. Gee recommended IV Mestinon. Id. Arterial blood gases showed respiratory alkalosis consistent with hyperventilation. Petitioner was admitted overnight for supportive measures. Id. Petitioner told Dr. Nguyen that she went on vacation about a week earlier and had a cold after she came back. She reported that she had not been sleeping well and was undergoing a significant amount of stress at home. She also had trouble swallowing and obvious stridor.[96] Id. Dr. Nguyen's impression was that petitioner had myasthenia gravis flare and she was put on BiPAP.[97] Id. Petitioner communicated by writing. Id. at 10.

On November 5, 2013, petitioner saw Dr. Jagmeet S. Mundi for evaluation of her upper airway. Id. at 12. Dr. Mundi put a fiberoptic tube down petitioner's upper airway. Id. at 13. Petitioner had evidence of significant laryngopharyngeal reflux disease secondary to esophageal reflux. She had hyperfunction of her true vocal cords including adduction of her vocal cords

[95] Protonix is "trademark for topical preparations of pantoprazole sodium." Dorland's at 1537. Pantoprazole sodium is "a proton pump inhibitor with properties similar to those of omeprazole, used in the treatment of erosive esophagitis associated with gastroesophageal reflux disease." Id. at 1371.
[96] Stridor is "a harsh, high-pitched breath sound such as the one often heard on inhalation with an acute laryngeal obstruction." Dorland's at 1785.
[97] BiPAP stands for "bilevel (biphasic) positive airway pressure." Med. Abbrev. at 65.

during phonation and inspiration, consistent with her history of inspiratory stridor. She did not have spasming of her vocal cords. The rest of her upper airway evaluation was within normal limits. Dr. Mundi notes petitioner was seronegative for myasthenia gravis, but there was strong suspicion for myasthenia, and inspiratory stridor is a known manifestation of myasthenia gravis. Id.

On November 7, 2013, petitioner was discharged to home and diagnosed with myasthenia gravis flare that was improved with IVIG, headache due to IVIG, and stridor related to myasthenia gravis flare which had resolved. Id. at 6. A CT of petitioner's chest showed no evidence of pulmonary embolism, aneurysm, or dissection; her lungs were clear. Id. at 7. On the day of discharge, petitioner was hemodynamically stable with no complaints. Id.

On December 2, 2013, petitioner saw Dr. Gee, who describes her as having atypical myasthenic syndrome, with seronegative biomarkers, but responds to pyridostigmine. Med. recs. Ex. 115, at 33. Thus far, tertiary care centers Mayo Clinic and UCLA have not determined another diagnosis. Dr. Gee states that IVIG therapy helped petitioner when she suffered a severe recent crisis[98] of weakness, with symptoms of upper airway stridor, respiratory distress, and exacerbation of gastroparesis. Id. Dr. Gee ordered Gamunex[99] IV solution to be infused every 30 days for six months. Id.

On February 4 and 5, 2014, petitioner underwent a 24-hour Holter monitor for palpitations because petitioner complained of dizziness, chest pains, and spots in her vision while running one mile and lifting weights. Med. recs. Ex. 115, at 36. According to the report, petitioner's baseline rhythm was normal, but she had sinus arrhythmia during nocturnal hours. Id. No arrhythmia was noted during the Holter monitor test and she had normal conduction intervals in sinus rhythm. Id.

On March 26, 2014, petitioner saw Dr. Gee again. Id. at 85. Petitioner appeared stable and showed a positive effect with IVIG. Id. Petitioner's physical examination was normal. Id. at 86.

On March 27, 2014, petitioner saw Dr. Daniel J. Wallace. Med. recs. Ex. 90, at 1. Petitioner reported that she had abnormal diffuse joint pain and she felt pain in her flank when she breathed deeply. Id. at 2. Dr. Wallace noted that on April 18, 2012, petitioner had an ANA titer of 1:160, which he marked as normal, and an ANA speckled pattern, which he also marked as normal. Id. Petitioner also reported that she had a feeding tube inserted in September 2013, was receiving IVIG treatment, and was using Mestinon every few hours for her condition. Id. Petitioner's autoimmune lab results showed high levels of CO_2, AST, and CPK. Id. at 6-7; Ex. 115, at 88-89. Dr. Wallace gave petitioner a neurology referral. Med. recs. Ex. 90, at 5.

On April 10, 2014, petitioner underwent a stress echocardiogram at Cardiology Consultants of Newport in Newport Beach, California. Med. recs. Ex. 115, at 90. The result was normal without evidence for stress-induced myocardial ischemia or dysrhythmia. Id.

[98] Myasthenic crisis is a "sudden development of dyspnea requiring respiratory support in myasthenia gravis; the crisis is usually transient, lasts several days, and is accompanied by fever." Dorland's at 431.
[99] Gamunex is "trademark for a preparation of immune globulin intravenous (human)." Dorland's at 757.

Petitioner's baseline echocardiogram showed mitral valve buckling without true prolapse and her left ventricular systolic function was normal. Id. She had mild mitral regurgitation. Id.

On April 15, 2014, petitioner had a test of her CPK which was normal. Id. at 91.

On September 3, 2014, petitioner saw Dr. Gee, reporting she was able to eat and swallow without bouts of weakness. Id. at 92. She found that the IVIG made a huge difference in her treatment. Id. Her physical examination was normal. Id. at 93-94.

On January 9, 2015, petitioner saw Dr. Gee, reporting some intermittent symptoms. Id. at 95. He noted petitioner was struggling. He counseled petitioner about taking Northera,[100] a new drug for autonomic dysfunction. He discussed her concerns over central sleep apnea and recommended a sleep study. Id. Her physical examination was normal. Id. at 97.

On January 29, 2015, petitioner saw Dr. Geoffrey L. Sheean,[101] a neurologist at Scripps Clinic Medical Group in La Jolla, California, complaining of myasthenia gravis (to which Dr. Sheean added a question mark) and dysautonomia. Med. recs. Ex. 116, at 1. Petitioner told Dr. Sheean she had flu vaccine two weeks before her rhabdomyolysis and, one week later, leg weakness neck extensor weakness, dysarthria, and dysphagia. Id. He notes petitioner did not have exertional dyspnea, ptosis,[102] diplopia, blurred vision, or dry mouth. Id. He also notes that CT and MRI chest scans did not show thymic[103] abnormality. Petitioner told Dr. Sheean that her reflexes disappeared and reappeared with prescriptions. Id. at 2. Dr. Sheean attributed petitioner's vertigo episodes to likely benign positional vertigo ("BPV"). A SPECT scan of petitioner's brain suggested vasculitis, but she did not have central nervous system symptoms. Id. An angiogram, which Dr. Sheean wondered was really an MRA, did not show vasculitis. Id.

On physical examination, petitioner had moderate symmetrical weakness in her face, tongue, jaw, neck extensors, proximal upper and lower limbs, girdle muscles, and distal upper limb muscles (intrinsic hand), with no post-activation improvement in strength. Id. Petitioner did not have atrophy or fasciculations. Her motor effort was very shaky, but she did not have any proprioceptive deficit. Her Romberg was negative. Her reflexes tested at zero or 1+, with no obvious post-activation facilitation, but petitioner would have a coughing spell after effort,

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[100] Northera contains droxidopa, which is a synthetic amino acid precursor of norepinephrine, used for the treatment of orthostatic dizziness, lightheadedness, and other illnesses. Northera, RXLIST, https://www.rxlist.com/northera-drug.htm#description (last visited Feb. 4, 2019).

[101] Dr. Sheean is from Australia. He is not board-certified in neurology in the US. The undersigned could not find him board-certified in other areas. Is My Doctor Board Certified?, AMERICAN BOARD OF MEDICAL SPECIALTIES, https://www.certificationmatters.org/find-my-doctor/?search=Geoffrey&lname=Sheean&state=CA&specialty=1012 (last visited Mar. 19, 2019). He graduated from the University of Queensland Faculty of Health Sciences in 1984 and is a research physician, specializing in electrophysiology. About Dr. Geoffrey Sheean, MD, U.S. NEWS HEALTH, https://health.usnews.com/doctors/geoffrey-sheean-984802 (last visited Mar. 19, 2019); Dr. Geoffrey Sheean, MD, HEALTHGRADES, https://www.healthgrades/com/physician/dr-geoffrey-sheean-2ktfw (last visited Mar. 19, 2019). Dr. Sheean was licensed to practice medicine in California on June 28, 2002. He did not identify having any board certifications to the Medical Board of California. The website information is self-reported. Medical Board of California, DCA SEARCH, https://search.dca.ca.gov/details/8002/A/79636/4aa0336fbf751a885ccecba6541c977c (last visited Mar. 19, 2019).

[102] Ptosis is "drooping of the upper eyelid." Dorland's at 1551.

[103] The thymus "is the site of production of T lymphocytes." Dorland's at 1925.

making testing difficult. Id. Petitioner's pupils were 5-6mm and poorly reactive. Id. at 3. She had mild ptosis bilaterally, left > right, which was non-fatiguable. A Cogan's lid sign[104] was absent. Extraocular movement ("EOM") showed normal pursuits with bilateral hypometric[105] horizontal saccades.[106] Her eyes were white. Her mouth was not dry. Id.

Dr. Sheean diagnosed petitioner with acute, progressive, severe dysautonomia involving the gastrointestinal, genitourinary, and cardiovascular systems, noting POTS and questioning Raynaud's, with possibly pupillary dysautonomia. Id. He also diagnosed petitioner with myasthenia, but he was unsure if it were myasthenia gravis ("MG") or Lambert-Eaton myasthenic syndrome (LEMS).[107] He entertained the possibility that petitioner could have mild residual myositis (CKs).

Dr. Sheean then considered four possible triggers: (1) flu vaccine leading to autoimmune myositis with rhabdomyolysis, triggering myasthenia gravis, and vaccine-triggered dysautonomia; (2) flu vaccine leading to autoimmune myositis with rhabdomyolysis, triggering LEMS; (3) acute viral myositis causing rhabdomyolysis, triggering MG+dysautonomia, or LEMS; or (4) acute autoimmune attack causing myositis (largely self-limiting) plus MG+dysautonomia or LEMS. Id.

Dr. Sheean stated:

> The severity of the dysautonomia argues against Lambert-Eaton myasthenic syndrome and that is supported by the very good response of the weakness to pyridostigmine. However, Lambert-Eaton myasthenic syndrome is still a plausible diagnosis and [since petitioner is] a young woman, would almost certainly be nonmalignant, not to mention that it has been more than 5 years since the onset. It is also important to distinguish myasthenia gravis from Lambert-Eaton myasthenic syndrome because of the option of thymectomy for myasthenia gravis, despite the absence of thymic enlargement.

Id.

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[104] Eric L. Singman et al., Use of the Cogan Lid Twitch to Identify Myasthenia Gravis, 31 J NEUROOPHTHALMOL 239 (2011). Of 117 patients evaluated, 24 had myasthenia gravis, of whom 18 had a positive lid twitch. Patients were instructed to look straight, up, down, and straight again. If the upper eyelids had a brief upward twitch, this indicated a positive Cogan Lid Twitch test.

[105] Hypometria is "dysmetria in which voluntary muscular movement falls short of reaching the intended goal." Dorland's at 903.

[106] Saccadic movement is "the quick movement of the eye in going from one fixation point to another." Dorland's at 1184.

[107] Lambert-Eaton myasthenia syndrome is "an autoimmune, myasthenialike syndrome caused by autoantibodies to the voltage-gated calcium channel (anna 1 antibodies) that interfere with the release of acetylcholine at the motor nerve terminal. Weakness usually affects the limbs, but ocular and bulbar muscles are spared; there is reduced muscle action potential on stimulation of its nerve but with repetitive stimulation it becomes augmented. It is often associated with oat-cell carcinoma of the lung." Dorland's at 1836.

Dr. Sheean further stated petitioner clearly needed to start immunosuppressants and recommended CellCept (mycophenolate mofetil)[108] for six months, with monthly monitoring of her complete blood cells and comprehensive metabolic panel. If CellCept failed, he would switch petitioner to tacrolimus.[109] After that, he would put her on rituximab.[110] He suggested she receive zoster vaccine. He suggested petitioner increase her IVIG because her myasthenia was still very symptomatic. He recommended repetitive nerve stimulation and short exercise testing for post-activation facilitation, which should distinguish between MG and LEMS. If not, petitioner would need stimulated single-fiber EMG. Id. He noted that voltage-gated calcium channel antibodies would indicate LEMS. Id. at 4. He ordered repeat testing of AChR antibodies and LRP4 antibodies.[111] If they were negative, he would repeat testing for MuSK. He would test for SSA/SSB antibodies for primary Sjögren's syndrome. In addition, he would check voltage-gated potassium channel antibodies for dysautonomia. Id.

On January 29, 2015, petitioner was tested for numerous antibodies, all of which were negative: SSA antibody and SSB antibody to test for Sjögren's; acetylcholine receptor modulating antibody; acetylcholine receptor blocking antibody; voltage-gated calcium channel antibody; voltage-gated potassium channel antibodies. Id. at 6-11.

On February 10, 2015, petitioner had repetitive nerve testing, which was normal. Id. at 12. Dr. Sheean reviewed her other tests results, all of which were negative: voltage-gated potassium channel antibodies, voltage-gated calcium channel antibodies, acetylcholine receptor antibodies, and Sjögren's antibodies. However, the laboratory had difficulty obtaining LRP4 antibodies. Dr. Sheean believed that the most likely form of myasthenia that petitioner had was myasthenia gravis Robinson Lambert-Eaton myasthenic syndrome. Id. Dr. Sheean thought it might be prudent to check to see if petitioner had MuSK antibodies, but there might be an option for thymectomy. A thymectomy would not help if she did have MuSK antibodies or Lambert-Eaton myasthenic syndrome. It also would not affect her autonomic neuropathy. Dr. Sheean thought petitioner's autonomic neuropathy could be a ganglionopathy perhaps due to antibodies to ganglionic acetylcholine receptors. Petitioner decided not to have a thymectomy and opted to proceed with immunosuppression and continue IVIG. Because petitioner had problems with aseptic meningitis, Dr. Sheean recommended premedication with Solu-Medrol to prevent infusion-related headaches. Id. Dr. Sheean recommended increasing IVIG dosage and starting CellCept. Id.

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[108] Mycophenolate mofetil is "an immunosuppressive agent used in conjunction with cyclosporine and corticosteroids to prevent rejection of allogeneic renal, hepatic, and cardiac transplants." Dorland's at 1216.

[109] Tacrolimus is "a macrolide suppressant of the calcineurin inhibitor group, derived from *Streptomyces tsukubaensis* and having actions similar to those of cyclosporine. Administered orally or intravenously to prevent rejection of organ transplants, especially liver." Dorland's at 1868.

[110] Rituximab is "a chimeric murine/human monoclonal antibody that binds the CD 20 antigen; used as an antineoplastic in the treatment of CD20-positive, B-cell non-Hodgkin lymphoma." Dorland's at 1650.

[111] LRP4 antibodies are lipoprotein receptor-related protein 4 antibodies. "Recently, the novel antigen, the low-density lipoprotein receptor-related protein 4 (LRP4), has been identified as a target for the autoantibodies in MG." Goichi Beck et al., Double Seronegative Myasthenia Gravis with Anti-LRP4 Antibodies Presenting with Dropped Head and Acute Respiratory Insufficiency, 55 INTERN MED 3361, 3361 (2016).

Also, on February 10, 2015, petitioner underwent another EMG/NCS. Id. at 13. Repetitive nerve stimulation was done on petitioner's right ulnar, accessory (trapezius) radial, and median nerves at rest and after exercise, in the absence of pyridostigmine. Petitioner did not have any abnormal decrement. A short exercise test was performed on the right radial and median nerves with no significant post-activation facilitation. Dr. Sheean's interpretation was, "At present, there is no indication of a neuromuscular junction disorder." He notes, however, that petitioner was receiving IVIG. Id.

On March 5, 2015, petitioner saw Dr. Gee. Med. recs. Ex. 140, at 2. Petitioner reported that she was doing better with the higher IVIG dose. Id. On physical examination, petitioner's motor skills, balance, and gait were intact. She did not have language deficits. Her deep tendon reflexes were preserved. Id. at 3. Dr. Gee recommended petitioner return every three to four months. Id. at 2.

On March 25, 2015, April 9, 2015, and August 3, 2015, petitioner saw Dr. Craig A. Salcido, an obstetrician-gynecologist, to seek consultation and treatment including a hysterectomy. Med. recs. Ex 173, at 1-5.

On July 17, 2015, petitioner saw Dr. Gee and discussed her intent to have a hysterectomy. Med. recs. Ex. 140, at 7. Petitioner appeared to be stable. Id. On physical examination, Dr. Gee noted petitioner did not have language deficits and her motor skills, balance, and gait were intact. Id. at 9.

On August 7, 2015, Dr. Salcido performed a power morcellation laparoscopic supracervical hysterectomy. Med. recs. Ex. 173, at 12; see also Ex. 140, at 11-16. Petitioner also received a pulmonary consultation from Dr. Robert Y. Goldberg on the day of her hysterectomy. Med. recs. Ex. 140, at 17. Dr. Goldberg assessed that petitioner "has a history of laryngeal spasm and myasthenia gravis." Id. His plan was to monitor petitioner postoperatively, monitor her respiratory status with vital capacities only, and "should respiratory status become compromised," he would have a low threshold to intubate. Id. Petitioner's EKG report, dated on the day of her procedure, showed that petitioner had normal sinus rhythm and a normal EKG. Med. recs. Ex 173, at 10. On August 10, 2015, Dr. Oscar H. Otanez's pathology report diagnosed "uterine tissue with benign glands and stroma; bilateral fallopian tubes with no significant histopathologic changes; negative for malignancy." Id. at 11.

On August 21, 2015, petitioner had a post-operation visit with Dr. Salcido, who noted that petitioner was "doing great," "very happy," and healing well. Id. at 12.

On June 15, 2016, during the second day of the hearing in this case, petitioner collapsed on the floor of the hearing room and was transported via EMS to the ED at MedStar Georgetown University Hospital. Med. recs. Ex. 179, at 1. Petitioner was "having difficulty breathing" and was nonverbal; she wrote for the EMTs that she could be treated only with Mestinon. Id. at 2. Petitioner reported that she "has a genetic disorder call[ed] (myasthenia gravis)." Id. Petitioner had "no swelling to tongue, mouth or face," and "she doesn't feel as though her throat is closing." Id.

At MedStar ED, Dr. Eric A. Glasser noted that petitioner was calm considering the amount of stridor she was having. Med. recs. Ex. 180, at 1. He requested a neurologic consultation. Id. at 2. Nurse Conway Luu noted petitioner's respiration at 1:20 p.m. to be diaphragmatic, gasping, grunting, labored, and shallow, with tachypnea[112] and use of accessory muscles. Id. at 25. Her respiratory pattern was described as regular. Id. Petitioner used furniture to assist herself in walking. Id. at 26. At 1:52 p.m., Nurse Luu noted petitioner was able to speak in full sentences, with slurred speech and wheezing to auscultation. Id. at 27. She had significant improvement in respirations, which were more relaxed. At 4:56 p.m., Nurse Luu noted petitioner had audible stridor bilaterally with shortness of breath. Id.

Dr. Brian Barry, a neurologist, spoke with petitioner who reported that she had an acute onset of shortness of breath associated with difficulty talking. Id. at 3. Petitioner said she had been recently sick with a sore throat, nasal congestion, mild weakness, and shortness of breath the prior night. However, she walked normally that morning. Around noon, she became acutely worse, and came to the ED. Petitioner denied problems with swallowing or diplopia but reported her lower extremities were weak. She said she had been unable to walk for the prior half-hour before arrival. Petitioner told Dr. Barry that she had never been intubated for myasthenia, and that she had been managed on BiPAP previously after she had similar symptoms in 2013. She said she took Mestinon at home as needed which greatly helped her symptoms, but she had not taken Mestinon that day. She said she was overdue for maintenance IVIG. Petitioner communicated with Dr. Barry through writing because she was not talking. She complained of fatigue. Id.

Dr. Barry did a physical examination of petitioner. Id. at 4. She was in mild distress and anxious. She did not have any wheezing. She had mild to moderate ptosis bilaterally which she could overcome spontaneously with upgaze and which did not worsen with fatigue. She had mild weakness in eyelid closure. She did not have nystagmus. Id. On motor examination, petitioner had normal bulk and tone. Id. at 5. She had bilateral drift and could sustain antigravity proximally for 1-2 seconds before the limb dropped. At maximum effort, her strength was 4/5 and symmetric. Her neck flexion and extension were 4/5. Her sensation was intact symmetrically to fine touch. She could do finger to nose bilaterally without dysmetria or tremor. Her reflexes were 2 everywhere except in the Achilles tendons where they were 1. Her big toes were downgoing. Dr. Barry's diagnosis was acute dyspnea. Her neurologic examination was significant for ptosis, weakness in eyelid closure, and diffuse weakness. However, she possibly had an element of effort dependence. Her negative inspiratory force ("NIF") was within normal and her arterial blood gases ("ABG") were reassuring. Id. Dr. Barry noted that petitioner's history was concerning for myasthenic exacerbation vs. crisis; however, her vital signs, NIF, and ABG were stable on admission. Id. at 5-6. Petitioner initially made "lots of upper airway noises with excellent respiratory effort," which are "inconsistent with MG weakness" and "more consistent with vocal cord dysfunction." Id. at 6. This resolved after a single dose of Mestinon.

[112] Tachypnea is "excessive rapidity of breathing." Dorland's at 1868.

Dr. Barry discussed petitioner's case at length with petitioner's physician Dr. Gee in California, who said a specialist at Scripps (which would be Dr. Sheean) diagnosed petitioner with seronegative MG with autonomic features. However, on multiple occasions, petitioner came to Dr. Gee with upper airway noises and shortness of breath. She was admitted to the hospital multiple times with this complaint. She was on monthly IVIG and was frequently admitted to the hospital with "low NIFs", but "no neuromuscular cause was believed to be associated with these spells." Id. Her myasthenia has been stable. Also, petitioner had "previously been assessed for non-organic dystonia attributed to a flu shot." Id.

On June 16, 2016, at 11:45 a.m., registered and licensed dietician Kelsie N. Hitesman noted that petitioner had "psychogenic stridor" and said she was currently unable to tolerate anything orally due to stridor and hesitance to swallow food, liquids, or medicine. Med. recs. Ex 183, at 90. Petitioner said she had been unable to take anything orally for two to three weeks and would take only oat milk via a feeding tube. Id. at 91. However, petitioner denied any weight changes in the last six months. Id.

On June 17, 2016, petitioner was discharged with a diagnosis of myasthenia gravis. Med. recs. Ex. 180, at 8. Petitioner was instructed to continue taking her medications as prescribed and to follow up with her outpatient neurologist within two weeks. Id. at 13.

On August 4, 2016, petitioner saw Dr. Sheean at Scripps to review her neurological condition and "provisional" diagnosis of myasthenia gravis. Med. recs. Ex 185, at 1. Dr. Sheean noted petitioner felt much better now that she was off oral contraceptives following her hysterectomy. She was now back working and did not have any more nocturnal shortness of breath. She was more active, which caused some muscle aching. She did not have evidence of joint hypermobility. Id. She had a continuing benefit from monthly IVIG, 2G per kg over three days, but it initially worsened her vertigo and autonomic symptoms (necessitating her using a feeding tube afterwards for two weeks). Id.

Petitioner reported right eyelid ptosis "now." Id. Dr. Sheean's physical examination of petitioner showed mild left ptosis. She also complained of poor visual tracking and diplopia, which was worse at the end of the day. Both symptoms responded to Mestinon. Petitioner said she was always dehydrated and had episodes of stridor that forceful inspiration, which occurred during pulmonary function tests, triggered. This resolved with IVIG. Her POTS was manageable. Petitioner was more troubled by weakness and fatigue, as well as her gastrointestinal autonomic symptoms. Dr. Sheean concluded petitioner likely had generalized, seronegative myasthenia gravis, especially with the addition of ptosis and diplopia. He discussed with petitioner her having a thymectomy, and testing for MuSK antibodies and LRP4 antibodies. He diagnosed petitioner with dysautonomia (gastroparesis and POTS), and episodic stridor which was likely laryngospasms that her forceful inspiration triggered. Id.

Dr. Sheean planned to retest petitioner for MG antibodies, including MuSK with an option for LRP4 antibodies if MuSK antibodies were negative. Id. at 2. If petitioner's MuSK antibodies were negative, he would repeat the single-fiber EMG with petitioner off IVIG, trying "to obtain better evidence of myasthenia gravis before considering thymectomy." Id. He would

consider a low dose of IVIG which might help both petitioner's dysautonomia and myasthenia. Id.

Dr. Sheean had petitioner undergo various tests. She had a high result for glutamic acid decarboxylase antibody ("GAD ab"), which was 22.2 when the normal range is 0.0-5.0U/ml. Id. at 3. Petitioner's creatine kinase ("CK") level was normal at 109 (reference range 26-192 units/L). Id. at 4. She was negative for Sjögren's antibodies (SSA and SSB antibodies). Id. at 8. She was normal for ceruloplasmin. Id. at 9. She was negative for MuSK antibody. Id. at 10.

On August 11, 2016, petitioner saw Dr. Gee. Med. recs. Ex 186, at 1. Dr. Gee noted petitioner had a positive GAD antibody, myasthenic syndrome, and muscle spasm. He counseled petitioner on the importance of the GAD antibody and the potential diagnosis of stiff person syndrome[113] and variant cerebellar ataxia. Id. He wrote orders for petitioner to test for GAD65, IA-2, and insulin autoantibody. Id. For review of systems, Dr. Gee wrote petitioner was negative for fatigue, dyspnea, wheezing gait disturbance, and psychiatric symptoms. Id. at 2. Upon physical examination, petitioner appeared well-nourished, alert, and oriented, with intact range of motion, no spasms, intact knowledge, no language deficits, normal attention span and concentration, fluent speech, no motor or sensory deficits, normal fine motor skills, and intact coordination, balance, and gait. She had preserved reflexes. Id.

On August 19, 2016, petitioner saw Dr. Sheean at Scripps to review her test results. Med. recs. Ex 188, at 1. The test results were as follows: (1) negative acetylcholine receptor antibodies and MuSK antibodies; (2) negative Mayo Clinic autonomic antibody panel--SSA, SSB, and Fodrin antibodies; (3) celiac haplotype risk <0.1x; and (4) positive GAD antibodies 22.2 (<5). Id. Petitioner reported that she experienced episodes of stridor and neck spasms in response to IVIG and questioned if she had stiff person syndrome. Id. Petitioner reported that she was clumsy and fell especially in the dark. Dr. Sheean thought her ataxia was sensory rather than cerebellar. She had a positive Romberg's sign and deafferentation[114] pseudoweakness with irregular motor effort on examination that she stabilized with visual fixation. The morning of her visit, all of petitioner's involuntary movements were slow, almost apraxic.[115] Her extraocular movements did not show nystagmus but seemed difficult for her to perform. Id.

Dr. Sheean assessed that petitioner has:

(1) Likely seronegative myasthenia gravis;
(2) Dysautonomia: cardiovascular (orthostatic) and gastrointestinal predominantly;
(3) Sensory ataxia, which did not appear to be cerebellar;

[113] Stiff person syndrome is "a condition of unknown etiology characterized by progressive fluctuating rigidity of axial and limb muscles in the absence of signs of cerebral and spinal cord disease but with continuous electromyographic activity; some cases have been linked to autoimmune conditions." Dorland's at 1849.
[114] Deafferentation is "the elimination or interruption of afferent nerve impulses, as by destruction of the afferent pathway." Dorland's at 473. The afferent pathway is "the nerve structures through which an impulse, especially a sensory impression, is conducted to the cerebral cortex." Id. at 1397.
[115] Apraxia is "loss of ability to carry out familiar, purposeful movements in the absence of paralysis or other motor or sensory impairment." Dorland's at 121.

(4) Cognitive impairment, likely autoimmune encephalopathy;
(5) Episodic muscle spasms, including laryngeal and cervical, possibly manifestations of stiff person syndrome, with accompanying anti-GAD antibodies; and
(6) Appears to have an incomplete DAME Syndrome (Dysautonomia, Autoimmune disease, Mast cell[116] activation disorder, Ehlers-Danlos syndrome[117]); absence of mast cell activation disorder.

Id. at 1-2. Dr. Sheean's plan was to "continue current IVIG regimen, recommend adding CellCept, 1G twice a day, advise against thymectomy for now." Id. at 2.

On August 14, 2016, petitioner had insulin autoantibody, GAD65 antibody, and IA-2 antibody tested. Med. recs. Ex. 189, at 1. Her insulin autoantibody result was normal. Her GAD65 antibody was high at 11 when the reference range was <5 IU/ml. Her IA-2 antibody result was normal. Id.

On January 16, 2017, petitioner saw Dr. Gee, complaining of weakness. Med. recs. Ex. 207, at 1. Dr. Gee stated petitioner was doing well and was stable. Id. He notes that her myasthenic syndrome resolved August 11, 2016. Id. He also notes that her POTS resolved on September 23, 2013. Id. He notes she had a prolonged QT interval.[118] Id. On physical examination, petitioner had intact range of motion and no spasms. Id. at 3. She was alert and oriented, with intact knowledge, normal attention span and concentration, fluent speech, and no language deficits. Id. She did not have any motor or sensory deficits, her fine motor skills were normal, her coordination, balance, and gait were intact, and she had preserved reflexes. Id.

On January 13, 2017, petitioner's GAD65 antibody was tested. Id. at 4. The result was high at 6 when the reference range was >5IU/ml. Id.

On May 2, 2017, petitioner had her GAD65 antibody tested. Med. recs. Ex 204, at 1. The result was high at 37 when the reference range was <5IU/ml. Id.

On July 17, 2017, petitioner saw Dr. Gee, stating she had been under stress, and had spasms and a bad rash along her trunk after taking Octagam.[119] Med. recs. Ex. 207, at 6. The review of systems and physical examination were normal. Id. at 7-8. Dr. Gee diagnosed petitioner with myasthenic syndrome, stiff person syndrome, POTS, positive GAD antibody, and

[116] Mast cell is "a type of connective tissue cell whose specific physiologic function remains unknown; it can elaborate basophilic, metachromatic, cytoplasmic granules that contain histamine and heparin in humans." Dorland's at 320.
[117] Ehlers-Danlos syndrome is "a group of inherited disorders of the connective tissue. The major manifestations include hyperextensible skin and joints, easy bruisability, friability of tissues with bleeding and poor wound healing, calcified subcutaneous spheroids, and pseudotumors." Dorland's at 1828.
[118] The QT interval is "in electrocardiography, the time from the beginning of the Q wave to the termination of the S wave, representing the time for ventricular depolarization." Dorland's at 951.
[119] Octagam is immune globulin intravenous preparation for treatment of primary humoral immunodeficiency. Common side effects include headache and nausea. Octagam, RXLIST, https://www.rxlist.com/octagam-side-effects-drug-center.htm (last visited Feb. 5, 2019).

pruritic rash. Id. at 8. Dr. Gee suggested petitioner try immunoglobulin, but not Octagam. Id. Dr. Gee's plan was to refer petitioner to dermatology for evaluation and treatment, request testing for immunoglobins, and renew carimune[120] and diazepam. Id.

On July 25, 2017, petitioner was tested for Immunoglobulin A, G, M, and E. Id. at 9. The results were normal. Id.

Medical Expert Reports

On October 22, 2013, petitioner filed the expert report of Dr. Lawrence Steinman. Ex. 65. Petitioner also filed on that date Dr. Steinman's CV. Ex. 66. On June 1, 2015, petitioner filed an updated CV, which Dr. Steinman dated May 23, 2015. Ex. 119. On January 13, 2016, petitioner filed another updated CV, which Dr. Steinman dated December 14, 2015. Ex. 143.[121] Dr. Steinman is board certified in neurology. Id. at 2. He is Professor of the Departments of Neurology and Neurological Sciences, Pediatrics and Genetics at Stanford University. Id. at 1. He is also incumbent of the G.A. Zimmermann Chair as Professor of Neurological Sciences, Neurology, and Pediatrics. Id. In 2004, he won the John M. Dystel Prize for Outstanding Contributions in Multiple Sclerosis Research, National MS Society and the American Academy of Neurology. Id. In 2009, he was elected to the Institute of Medicine, renamed in 2015 as the National Academy of Medicine. Id. In 2011, he won the Charcot Prize for Lifetime Achievement in MS Research—International Federation of MS Societies. Id. In 2015, he was elected to the National Academy of Sciences.[122] Id. Dr. Steinman has 38 patents. Id. at 2-3. He is associate editor of the journal Neurobiology of Disease. Id. at 4.

Dr. Steinman had been on the board of directors of Centocor from 1991-99 when it was sold to Johnson and Johnson.[123] Id. He was the founder advisor of Neurocrine Biosciences from 1992-2005, and on the board of directors from 2001-2005. He was on the scientific advisory board ("SAB") of Roche Biosciences from 1998-2002. He was the founder of Bayhill Therapeutics, head of its SAB from 2011, and a member of the board of directors. He was the founder and a board member of Atreca, Cardinal Therapeutics, and Tolerion. He was the

[120] CSL Behring, the manufacturer of Carimune, an immune globulin intravenous product, discontinued its production in the third quarter of 2018. CSL Behring to Discontinue Production of Carimune NF, IMMUNE DEFICIENCY FOUNDATION, https://primaryimmune.org/news/csl-behring-discontinue-production-carimune-nf (last visited Feb. 5, 2019).

[121] Posted on the internet in PDF format is the latest version of his CV, dated July 14, 2018. Rather than the 514 articles Dr. Steinman authored or co-authored listed on his December 14, 2015 CV, his 2018 CV lists 566 articles. Curriculum Vitae Lawrence Steinman, MD, STANFORD PROFILES, https://cap.stanford.edu/profiles/viewCV?facultyId=3784.Lawrence_Steinman (last visited Mar. 8, 2019).

[122] The National Academy of Sciences lists Dr. Steinman's research interests as the pathogenesis of multiple sclerosis and related neuroinflammatory diseases; development of antigen-specific tolerance therapy for autoimmune diseases where the autoantigen is clearly defined, particularly type 1 diabetes and neuromyelitis optica; and the pathogenesis and therapy of diseases in which amyloid structures are central in pathogenesis, including Huntington's disease and Alzheimer's. Member Directory. Lawrence Steinman, NATIONAL ACADEMY OF SCIENCES, www.nasonline.org/member-directory/members/14142.html (last visited Mar. 8, 2019).

[123] Johnson & Johnson announced its purchase of Centocor for $4.9 billion in stock on July 21, 1999. Johnson & Johnson to Acquire Centocor, THE NEW YORK TIMES (July 22, 1999), https://www.nytimes.com/1999/07/22/business/johnson-johnson-to-acquire-centocor.html.

founder and head of the SAB of Transparency Life Sciences. He was on the SAB of Receptos[124] starting in 2012 until the date of this CV, May 23, 2015. Dr. Steinman has also been affiliated with Janssen Biotech, Inc., Peptimmune, Inc., Garnet Biotherapeutics, Inc., Neurion Pharmaceuticals, Inc., Provid Pharmaceuticals, Inc., Biocon Limited, Vaccinex, Inc., Horizon Pharmaceutical LLC, KAHR Medical Ltd., BioAtla, LLC, Sequenta, Inc., Syapse, Bionure, Inc., and Applied Therapeutics, Inc.[125]

Dr. Steinman's opinion is that petitioner developed profound neurologic and muscle disturbances with rhabdomyolysis followed by serious autoimmune dysautonomia, a type of inflammatory autoimmune neuropathy, whose onset was approximately September 3, 2009, about 10 days after she received flu vaccine. Ex. 65, at 1. He states flu vaccine caused her rhabdomyolysis and autoimmune dysautonomia due to molecular mimicry between flu vaccine and myelin proteins ("MBP") leading to nervous system inflammation. Id. He says there are MBP sequences with which various viruses, including influenza virus A, can cross-react. Id. at 11.

Dr. Steinman states that portions of the autonomic nervous system are myelinated. Id. at 14. Thus, inflammation directed to myelin can affect autonomic function. Id. at 15. He writes activation of petitioner's innate immune system exacerbated a chain reaction of autoimmune reactions, including many serologic indications that she has lupus in addition to dysautonomia. Id. at 17. Dr. Steinman does not mention the fact that all the serologic testing of petitioner resulted in the conclusion that she does not and never did have lupus. He also focuses intently on how flu vaccine causes Guillain-Barré syndrome ("GBS"), but he does not mention the fact that doctors tested petitioner for GBS and concluded she did not have it. Dr. Steinman states:

> Activation of innate immunity and adaptive immunity to influenza vaccine components is the more likely reason that immunity to myelin occurred in [petitioner's] case. The autonomic nervous system is myelinated in many of its anatomic locations, and autonomic dysautonomia is at times a major feature of inflammatory demyelinating neuropathy and at times GBS. Autoimmune neuropathies with dysautonomia are often the result of immunity directed to the ganglionic AChR. Once inflammation is induced to myelin proteins via molecular mimicry to the influenza vaccine, various pathogenic reactions occur including autoimmune ganglionopathy.

Id. at 19.

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[124] Celgene bought Receptos for $7.2 billion in cash, announced July 14, 2015. Celgene to Acquire Receptos, Advancing Leadership in Immune-Inflammatory Diseases, CELGENE (July 14, 2015), https://ir.celgene.com/press-releases/press-release-details/2015/Celgene-to-Acquire-Receptos-Advancing-Leadership-in-Immune-Inflammatory-Diseases/default.aspx.

[125] Executive Profile. Lawrence Steinman, Co-Founder and Member of Scientific Advisory Board, Nuon Therapeutics, Inc., BLOOMBERG, (March 8, 2019), https://www.bloomberg.com/research/stocks/private/person.asp?personId=1153833&privcapId=35786930.

Dr. Steinman states petitioner had autoimmune autonomic ganglionopathy ("AAG"). Id. at 20. He reflects that no one tested petitioner for antibody to ganglionic acetylcholine receptor, which would appear in 50% of patients with autoimmune ganglionopathy. Id. at 21. However, he states whether petitioner tested positive or negative for antibody to ganglionic acetylcholine receptor would not "undermine" his conclusion. But if petitioner did have a positive result on testing for antibody to ganglionic AChR, that would be an important finding. Id. Dr. Steinman notes this is a "complex case," but he has a "high degree of medical certainty" that flu vaccine caused petitioner's rhabdomyolysis and subsequent autoimmune dysautonomia due to an autoimmune inflammatory neuropathy. Id.

On April 25, 2014, respondent filed the expert report of Dr. Peter D. Donofrio. Ex. B.[126] Respondent also filed on that date Dr. Donofrio's CV. Ex. C. Dr. Donofrio is board certified in neurology, electrodiagnostic medicine, and internal medicine. Id. at 2. At the time of the filing of his expert report, he was Director and Professor of Neurology at the Vanderbilt University School of Medicine, Neuromuscular Division. Id. He was a fellow with the American Academy of Neurology, and a member of the following associations: American Association of Electromyography and Electrodiagnosis, the American Neurological Association, the American Medical Association, and the Tennessee Neurological Association. Id. at 3. He authored or co-authored 86 articles (id., at 12-20), 12 book chapters (id., at 20-21), 83 abstracts (id., at 21-28), and 13 monographs (id., at 28-29).

Dr. Donofrio notes that petitioner's medical records show that her jerking and dystonic posturing improved when a doctor distracted her and her movements tended to fluctuate during the day. Id. at 7. She also had a speech aberration. Both the senior neurology resident and the neurology attending physician at Johns Hopkins University in early October 2009 had a strong suspicion that petitioner had a non-physiologic disorder. She was able to run, but not to walk forward unless she put her hand on her thigh. Her speech worsened when she rose from a chair, but her blood pressure did not change. Id. Dr. Donofrio notes that petitioner's expert Dr. Steinman ignored all these anomalies in petitioner's presentation and failed to explain how they are consistent with his diagnosis in his expert report. Dr. Donofrio states he is unaware of any neurologic condition that would manifest the way petitioner's condition manifested. Id. Petitioner's head CT scans, brain MRIs with and without contrast, and PET CT scan were normal and did not show brain pathology that would cause a movement disorder or autonomic dysfunction. Dr. Donofrio states petitioner's movement disorder cannot be related to any form of dysautonomia. Id.

As for petitioner having POTS, testing did not show petitioner had a significant change in pulse or blood pressure during up-tilt testing to substantiate the diagnosis of either POTS or orthostatic hypotension. Id. at 7-8. To diagnose POTS, petitioner should have had a 30-point rise in her pulse within the first 10 minutes of tilting or a pulse greater than 120 within the first 10 minutes in the setting of little change in blood pressure. Id. at 8. In addition, petitioner did not have blood pressure features of an autonomic neuropathy because she should have had a

[126] On August 15, 2013, respondent filed Exhibit A (A1 to A5). It is a CD of video files originally in Final Cut Pro format (petitioner's Exhibit 58), which respondent converted to a format viewable in Windows Media Player.

profound drop in blood pressure when she stood or was tilted up associated with little change in her pulse. Id.

Dr. Donofrio stated if petitioner had dysautonomia from autoimmune ganglionopathy, she should have had clinical symptoms and signs of a global autonomic disorder, i.e., sluggish pupillary responses, dry eyes and mouth, slow pulse, documented orthostatic hypotension, gastric bloating, constipation, urinary retention, sexual dysfunction, and dry skin. Id. These findings are not in her medical records. Dr. Donofrio notes that in November 2013, petitioner denied any autonomic features, including nausea, vomiting, abdominal pain, and dizziness. Dr. Donofrio stated petitioner did not have pulse or blood pressure features of either POTS or orthostatic hypotension. Id.

Dr. Donofrio observes that petitioner had a pre-existing history of recurrent bronchitis since February 2009 and a probable viral illness in early September 2009 because she had sore throat, fever, chills, muscle aches, and fatigue. Id. She was thought to have viral myositis, but continued training for a 5K race. In her September 12-14, 2009 hospitalization, the diagnosis was viral myositis because her creatine kinase values were very high at 12,000, although her neurologic examination was normal including normal deep tendon reflexes. Because petitioner had normal deep tendon reflexes and normal strength, she was not diagnosed with GBS. Moreover, GBS rarely causes elevated creatine kinase levels and, when it does, the elevation is mild and not 12,000. Petitioner also had normal reflexes when she went to Johns Hopkins University on October 3, 2009. Because of her neurological evaluations at Inova Loudon and Johns Hopkins, Dr. Donofrio does not believe petitioner had GBS. Id.

Dr. Donofrio thinks petitioner's viral illness rather than her flu vaccination caused her rhabdomyolysis since the vaccination occurred on August 23, 2009, but her symptoms did not develop until early September 2009, about 10 days later. Id. He notes that rhabdomyolysis is common in athletes and people who participate in regular, strenuous physical activity. Petitioner ran daily to prepare for a race. Id. Dr. Donofrio says that even if the vaccination caused rhabdomyolysis, it would not predispose petitioner to an autoimmune dysautonomia. Id. at 8-9. The muscle breakdown causing elevated CK levels after a viral illness is due to the virus's effect on muscle rather than to autoimmune disease. Id. at 9.

On August 14, 2014, petitioner filed Dr. Steinman's first supplemental expert report. Ex. 92. He states dysautonomia or autonomic dysfunction is a broad term describing any disease or malfunction of the autonomic nervous system ("ANS"). Id. at 1. The ANS controls numerous bodily functions, e.g., heart rate, blood pressure, digestive tract peristalsis, and sweating. Because dysregulation of the ANS can produce apparent malfunction of the organs it regulates, patients with dysautonomia often present with numerous, seemingly-unrelated maladies. Id. He lists the types of dysautonomia: POTS, inappropriate sinus tachycardia ("ITS"), vasovagal syncope, pure autonomic failure, neurocardiogenic syncope ("NCS"), neutrally-mediated hypotension ("NMH"), orthostatic hypotension, orthostatic hypertension, autonomic instability, and lesser-known disorders such as cerebral salt-wasting syndrome. Id. He lists some of the symptoms of patients with dysautonomia: excessive fatigue; excessive thirst (polydipsia); lightheadedness, dizziness, or vertigo; anxiety or panic that is not mentally induced; rapid or

slow heart rate; orthostatic hypotension sometimes resulting in syncope (fainting); blood pressure fluctuation; difficulty breathing or swallowing; gastroparesis (delayed gastric emptying) with associated nausea, acid reflux, and vomiting; and activity- and exercise-induced heat intolerance. Id. at 2.

Other symptoms frequently associated with dysautonomia are: headaches, pallor, malaise, facial flushing, salt craving, mydriasis (abnormal dilation of pupils), constipation, diarrhea, nausea, acid reflux, visual disturbances, numbness, nerve pain, chest pains, and sometimes loss of consciousness and seizures. Id. Petitioner was diagnosed with neurocardiogenic syncope, syncope and dystonia, POTS, gastroparesis, etc. Id. Dr. Steinman counted 17 treating physicians who acknowledged petitioner had conditions or symptoms consistent with autoimmune dysautonomia. Id.

Dr. Steinman chooses to ignore petitioner's viral illness which she had in early September 2009 because "we do not know the exact nature of the alleged illness." Id. at 6. He continues, "I do not see any objective evidence of an infection, virus or any confirmed microbiologic diagnosis of a virus or bacteria." Id. However, even if petitioner did have a virus, Dr. Steinman says, the virus and the flu vaccine could have acted in combination, meaning the vaccine would have been a substantial factor, i.e., but for the vaccination, petitioner would never have had these issues. Id. Dr. Steinman disagrees with Dr. Donofrio that petitioner's training for a 5K race was sufficient exertion to cause rhabdomyolysis. Id. Dr. Steinman says that he was not trying to opine in his initial report that petitioner's rhabdomyolysis caused her neurological symptoms, i.e., autoimmune inflammatory neuropathy. Id. at 7.

Dr. Steinman thinks that his discussion of data concerning GBS in his initial expert report is relevant to petitioner even though petitioner did not have GBS. Id. His opinion remains that flu vaccine caused petitioner's rhabdomyolysis and autoimmune dysautonomia due to an autoimmune inflammatory neuropathy, i.e., ganglionopathy. Id. at 8.

On August 14, 2014, petitioner filed two charts Dr. Steinman prepared: (1) Exhibit 93, entitled "Analysis of Doctors Who Agree Autoimmune Dysautonomia;" listing 20 doctors[127] over seven pages, and (2) Exhibit 94, entitled "Symptoms Relevant to Opinion of Autoimmune Dysautonomia," listing over 100 symptoms[128] on 11 pages. Out of those more than 100

[127] The doctors Dr. Steinman listed on the chart who purportedly agree petitioner has autoimmune dysautonomia are: Dr. Atiga, Dr. Buttar, Dr. Cintron, Dr. Ghassemi, Dr. Grubb, Dr. Ho, Dr. Naya, Dr. Lyden, Dr. Olek, Dr. Tannenbaum, Dr. Tang, Dr. Tran, Dr. Urrutia, Dr. Sharrief, Dr. Virmani, Dr. Wallace, Dr. Waxman, Dr. Wilkinson, Dr. Wohlman, and Dr. Yan-Go. Ex. 93, at 1-7. Only five of these doctors are neurologists: Dr. Cintron, Dr. Lyden, Dr. Olek, Dr. Sharrief, and Dr. Yan-Go.

[128] The symptoms Dr. Steinman finds relevant to the diagnosis of autoimmune dysautonomia are: syncope, near syncope, shaking, weakness, dizziness, lightheadedness, increased respiratory rate, cough, fatigue, feeling weak and tired, rhabdomyolysis, leukocytosis, recurrent respiratory issues, elevated liver function tests, elevated creatine kinase, heart murmur, shortness of breath, muscle weakness, tingling in hands and feet, passing out while sitting or standing, fainting or convulsions (sometimes after eating), lower extremity weakness, headaches, neck/joint pains, generalized tremors, worsening of extremity weakness, difficulty ambulating, easily winded, feeling legs would buckle from lack of strength, trouble concentrating, stuttering speech, shooting and tingling pains in lower extremities, walking and performing motor functions for 15 minutes in the morning before tremors would set in, syncope after meals, hot flashes vs. fever, symptoms such as tremors that did not start right away each morning but

symptoms, Dr. Steinman finds irrelevant to the diagnosis of autoimmune dysautonomia only five symptoms: hyperventilation, talking in one-word answers, cold symptoms (body aches, sore throat, not feeling well), pseudoseizures, and cold spots in the back of her head.

On August 28, 2014, petitioner filed Dr. Steinman's second supplemental expert report. Ex. 108. He reiterates that petitioner had autoimmune dysautonomia due to autoimmune neuropathy as a result of flu vaccination on August 23, 2009. Id. at 1. He states, "Autoimmune inflammatory neuropathy is also known as Guillain Barre Syndrome." Id. He observes that GBS may have autonomic nervous system manifestations. Dr. Steinman then proceeds to describe the various manifestations of autonomic changes in someone who has GBS. Id. He states "dysautonomia is a variant of GBS." Id. at 2. He admits he concludes petitioner had GBS by "inference," i.e., he infers since she had autonomic dysfunction, she had GBS. Id.

On January 13, 2015, respondent filed the expert report of Dr. Eric Lancaster, a neurologist. Ex. H. Respondent filed Dr. Lancaster's CV as Exhibit I. Respondent filed an updated CV of Dr. Lancaster on May 6, 2016. It is dated April 18, 2016. Ex. KKK. He states in his CV that he has expertise in antibody-mediated neurological disorders. Id. at 1. He is based in the Center for Autoimmune Neurology at the University of Pennsylvania. Id. He is Assistant

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30 minutes after waking up or soon after eating and then being unable to move her limbs, spastic jerking limb movements, dystonic posturing, jerking movement of the entire body while walking, normal reflexes and strength, stuttering and halting speech which would disappear upon whispering, inability to hold a cup of water in her hand without spilling, trouble walking, dysarthria, abnormal gait, ascending bilateral lower leg weakness, dysphonia, episodes of uncontrollable blinking, inappropriate laughter, photophobia, tinnitus, syncopal episodes associated with eating, ataxic waddling gait, foot tapping, head bobbing, jerky vertical head tremor varying according to the level of exertion, ability to walk backward and sideways but not forward without abnormal gait and head bobbing, speech issues relieved by placing her hand on her chin, touching her anterior left thigh or fastening a belt around her leg to allow her to walk normally, memory difficulties, concentration difficulties, labored breathing, back to back seizure/contractures, gross motor deficits, loss of coordination, inability to ambulate, inability to articulate with dysphonia, absence of fine motor skills, ability to run an 8K race two days prior to rapid deterioration, inability to keep food down without vomiting, changing speech tones and a foreign accent (British or Australian), easier to breathe while running than at rest, walking straighter if she looked to the left, heartbeat increasing to 200 while exercising, symptoms improving with exercise, overheating resulting in syncopal episodes, exercising excessively to prevent syncope, difficulty walking but no problem running except would sometimes pass out after running, frequent seizures (about 60 per day) without losing consciousness, difficulty reading, difficulty recalling/strategizing, trouble with noise/lights which exacerbated her problem with talking and caused stuttering, loud noises causing seizure, inability to do math, directional confusion, eating making her pass out, arm and face muscle contractions while speaking, inability to focus on words on a page, inability to read and comprehend, weakened cognitive skills, stress exacerbating her symptoms, sinus tachycardia, normal heart rate and blood pressure response to exercise, post-exercise syncope secondary to neurocardiogenic hypotension, unusual form or neurocardiogenic syncope that improved with exercise because exercise would increase her sympathetic tone, tilt-table test showing that petitioner's clinical symptoms were reproduced and worsened in upright tilt position by provocation with nitroglycerin, Dr. Atiga's suggestion of cerebral syncope as dysregulation of cerebral blood flow to upright posture especially with orthostatic stress, not eating or drinking but vomiting and dry heaving, facial weakness, heat intolerance, postural orthostatic tachycardia syndrome (POTS), postprandial hypotension, poor exercise tolerance but feeling best when exercising vigorously, SPECT brain scan showing reduction in watershed bilaterally and decreased perfusion in right thalamus, Raynaud's of the brain, reaction to central nervous system anti-depressant drugs and SSRIs, gastrojejunostomy tube, controlling breathing with extreme concentration, tremors resolving each morning to apply makeup but later unable to speak or move limbs, spastic jerking, dystonic posturing, uncontrollable blinking, and absence of fine motor skills. Ex. 94, at 1-11. The undersigned is at a loss to understand why Dr. Steinman included normal reflexes, normal strength, and normal heart rate and blood pressure responses to exercise as symptoms of autoimmune dysautonomia.

Professor of Neurology at The University of Pennsylvania. Id. He is board certified in neurology, electrodiagnostic medicine (scoring in the top 10 percent of the examination), and neuromuscular medicine. Id. at 2. Dr. Lancaster's thesis for his Ph.D. concerned the physiology of the autonomic nervous system. Id.

Dr. Lancaster is a member of the following associations: Society for Neuroscience, the American Academy of Neurology, and the American Association of Neuromuscular and Electrodiagnostic Medicine. Id. He has authored or co-authored 32 articles. Id. at 3-5.

Dr. Lancaster reviewed videos filmed by Generation Rescue (Ex. 58), 20/20 news reports, and petitioner's medical records. Ex. H, at 1. Dr. Lancaster notes that on October 17, 2009, petitioner completed the Brain Aneurysm Awareness 8K run in 56 minutes and 13 seconds (8K is almost 5 miles). Id. at 4. He says the diagnosis of petitioner's having an abnormal qEEG done on October 22, 2009 was illogical because it said petitioner had absence epilepsy over one part of her brain, but absence epilepsy is a genetic condition and would not suddenly occur at petitioner's age. Dr. Lancaster does not believe this study validly measured petitioner's brain. Id. at 5. Referring to Dr. Buttar's test conclusion that petitioner had heavy metal poisoning on October 27, 2009, Dr. Lancaster states it is highly improbable that a single flu vaccination caused heavy metal poisoning. Id. Moreover, petitioner's test for mercury was normal just a week before on October 20, 2009. Id. On November 9, 2009, a news story by vactruth.com reported that Dr. Buttar had cured petitioner. Id. Dr. Lancaster notes that a similar report appeared on http://www.ageofautism.com/2009/11/nfl-cheerlreader-disabled-by-2009-flu-shot-on-road-to-recovery.html. Id.

Dr. Lancaster notes that on November 18, 2009, Dr. Buttar saw petitioner and recorded that her cognitive functioning had improved, and she could reportedly read one sentence at a time but without comprehension. Id. at 5-6. Petitioner had a complex series of speech symptoms, including losing all speech on November 3, 2009. Id. at 6. Petitioner spoke with an accent afterward. Dr. Buttar diagnosed her with mercury toxicity and encephalopathy. Petitioner was on chelation therapy. Id. On December 1, 2009, Dr. Buttar noted petitioner could not do math or read. Dr. Lancaster does not believe petitioner's lab results establish Dr. Buttar's diagnoses. Id.

On July 15, 2010, petitioner underwent a tilt-table test. Id. at 7. She was tilted upright and developed slurred speech progressing to a total inability to speak for more than 15 minutes. Id. Her blood pressure and heart rate were normal the entire time. Id. at 7-8. When petitioner was laid flat, her dystonia resolved immediately, but her speech remained slurred for nearly an hour. Id. at 8. The tilt-table report concludes petitioner had normal heart rate and blood pressure response. Id. On September 9, 2010, petitioner underwent another tilt-table test. Id. Petitioner's blood pressure remained stable, but one data point showed elevated pulse. Her response to deep breathing was normal. The diagnosis was grade 2 POTS. Id.

Discussing videos filed into evidence, under the subheading "October 17, 2009 videos," Dr. Lancaster describes a video (#00071W.mov) showing petitioner complaining of having no

thoughts and speaking in a slow, labored voice. Id. at 11. Dr. Lancaster states this pattern of speech does not correspond to any neurological disorder in his experience. Id.

Dr. Lancaster notes in another video (#409_0239_01), petitioner says she cannot drink water without triggering convulsions. Id. at 12. Dr. Lancaster states this complaint does not correspond to a neurological disorder in his experience. Id.

In another video (#409_0240_01), petitioner was preparing to run a race while giving an interview to channel 5. She reports she cannot talk or walk normally and the only thing she can do is run. She says when she runs, her voice returns. She wanted to warn people about what was in vaccines. Id.

In another video (#409_022_02), petitioner discusses how all her symptoms resolve when she is running. Id.

In another video (#409_0245_01), petitioner drinks a large glass of water and eats several large bites of food very quickly. She has no problem swallowing. Then she immediately drops her head and has a strained voice. She is assisted to a couch and lies on her side, continuing to eat and feed herself while jerking her head with her eyes shut. Even with the jerking, she chews and swallows the food without difficulty. Id. She also demonstrates good fine motor control. Id. at 13. She begins to suck on a lollipop without difficulty, but then reports it is hard to breathe. Dr. Lancaster comments that an epileptic seizure with altered consciousness and generalized shaking would have prevented petitioner from sucking the lollipop. Id.

Another video (#409_252_01) shows petitioner lying on the couch, being fed while reporting difficulty moving her tongue. Her then-husband says that petitioner's moving the food around with her tongue causes her to seize. Dr. Lancaster notes this is inconsistent with her claim her tongue is paralyzed. The event appears to Dr. Lancaster to be most consistent with a psychogenic, non-epileptic seizure-like movement due to bilateral jerking, but preserved consciousness. Id.

In another video (#409_254_01), petitioner is lying on her back, speaking with a slurred pattern, and shaking, which Dr. Lancaster interprets as most consistent with a psychogenic event.

In another interview (video #409_255_01), petitioner is sitting on a couch and speaking with a slurred voice again. Id. While she is being interviewed, petitioner has a couple of episodes of head jerking which her then-husband says are seizures. Petitioner discusses how prominent her seizures were as an early symptom after vaccination. Occasionally, she speaks with a British accent. The severity of petitioner's speech problem fluctuates considerably during the interview. Petitioner claims she had severe leg weakness and could not walk. Id. In another video, petitioner continues her interview, and her speech is almost normal. When she whispers, she has excessive facial movement. She says thinking up words is difficult. She claims reading can sometimes trigger a seizure. She says that she was angry when someone in the hospital told her she was crazy. Id.

Dr. Lancaster describes the next video (#409_0257_02), which is a continuation of petitioner's recounting her initial illness, describing seizure-like episodes, ability to walk

backward but not forward, and periods of being able to walk but not talk or talk but not walk. She says she can relieve vocal problems by putting her hand on her chin. Id. Dr. Lancaster comments that a real autoimmune brain disease causing neurological deficits would not have resolution and recurrence within seconds and would not respond to sensory tricks.

In another video (#409_0257_03), petitioner states all her symptoms immediately recur when she stops running, which Dr. Lancaster notes is also inconsistent with an autoimmune brain disease. Petitioner says in the video that she has convulsions and blacking out when she stops running. Id.

Under the subheading "October 18, 2009 videos," Dr. Lancaster describes three videos (#409_0272_01, #409_0272_02, and #409_0272_03) in which petitioner discusses injuries from the vaccination while using a computer. Id. at 14. Petitioner speaks in a strained and unusual way and then sucks on a lollipop, swallowing without apparent difficulty. She also has no difficulty using her hands to operate the computer, pet her dog, or suck the lollipop. Dr. Lancaster finds it interesting that petitioner could operate a computer because, in other videos, she reported profound trouble reading or thinking. Id.

In another video (#409_0278_01), petitioner is taking liquid medicine from a spoon, swallowing pills, then drinking liquid several times, all without difficulty. Id. At 4:22 p.m., she has a non-epileptic (psychogenic) seizure-like event. Dr. Lancaster notes that during the event, petitioner's body was shaking but she was able to put her drink down properly. He notes that her eyes were closed during the event, which he calls a predictive factor for a non-epileptic event. He comments that petitioner carefully positioned herself to avoid injury during the event. Id.

In three videos (#409_0278_03, #409_0279_01, and #409_0279_02), petitioner is lying on a couch and speaking with a slurred voice. Plans are to film her visit to Dr. Buttar's clinic in North Carolina. In video #409_0279_01, petitioner's then-husband is typing on a laptop next to petitioner and states petitioner learned about sensory tricks on the Mayo Clinic website for dystonia after a physical therapist mentioned the diagnosis of dystonia. Id.

In another video (#409_0283_01), petitioner discusses how the back of her head feels cold and she cannot make it warm. Id. at 15. Later, petitioner reports burning in her neck and similar burning ("on fire") while undergoing an MRI. She later explains she believes this burning is due to her having mercury in her system. She becomes very upset and then has head shaking while saying repeatedly, "It burns." Her then-husband helps her lie down, while she repeatedly says, "It burns," clenches her fists, and has someone support her jaw. She says the burning is like "acid in the brain." Her then-husband says she had periods of two hours of involuntary laughing. Dr. Lancaster explains that mercury is not paramagnetic and, thus, a magnetic field from an MRI would not affect her this way, particularly if mercury were dissolved in the human body. Id.

In another video (#409_0298_01), petitioner's then-husband describes petitioner having a seizure-like event. At Johns Hopkins, she had Ativan and then, when she woke, she spoke perfectly normally. She later had a miraculous recovery of her strength, doing 20 laps and a little dance in her room, but then suddenly went into "big time payback seizures." Id. A physical

therapist thought she had dystonia and mentioned medication and sensory tricks. Id. at 16. Petitioner seemed to get her speech and walking back with benzodiazepines. She would have "seizures" with any kind of stimuli, e.g., a dog barking or a plane flying overhead, or if more than one thing was occurring at a time. She attributed these "seizures" to benzodiazepines. She spoke to someone on the phone who thought she had an immune problem and that the benzodiazepines were treating it. Dr. Lancaster comments that there is no medical basis for opining that benzodiazepines treat an autoimmune disease. Moreover, Dr. Lancaster thought it extremely unlikely that the events of dog barking or planes flying overhead would trigger epileptic seizures. His opinion is that petitioner was having psychogenic, non-epileptic events and not epileptic seizures. Id.

Another video (#409_0299_01) shows a phone conversation with Dr. Buttar, who had yet to meet petitioner. He said he wanted to cool her system down and said he could treat it. He wanted her to come to a controlled environment to get supplements and endorsed hyperbaric therapy to treat brain inflammation. He endorsed IV fluids, but not IVIG which he thought would be detrimental. Id.

Under the subheading "October 19, 2009 videos," is a video of petitioner arriving at Dr. Buttar's clinic. Petitioner is lying down and communicating with gestures, but not talking. Id. Petitioner has rhythmic head shaking when a nurse asks her a question. Id. at 16-17. Petitioner covers her face with her hands. Dr. Lancaster comments this does not look like epilepsy but is another non-epileptic psychogenic event. Id. at 17.

In another video (#409_0314_01), petitioner's then-husband and Stan Kurtz discuss how anything, such as eating or seeing a floor pattern, could trigger petitioner to seize. Dr. Lancaster states that eating or seeing a floor pattern as a trigger for seizures supports the diagnosis of psychogenic events rather than epileptic seizures. Id.

Video #409_0325_01 shows petitioner being spoon-fed. She then stares. Stan Kurtz says, "She's seizing." Petitioner responds somewhat to a hand near her eye and then hyperventilates a bit when the event stops. Dr. Lancaster comments this event was most consistent with another psychogenic, non-epileptic event.

In video #409_0326_01, petitioner makes retching noises but does not vomit. She clenches her arms and has another shaking event. Dr. Buttar arrives. Petitioner lies on the bed with her eyes closed as Dr. Buttar speaks. He plans to insert an IV. Stan Kurtz states that when petitioner is "in this state, any activity can set her off." She has head shaking after she is asked to squeeze Dr. Buttar's hand. Dr. Lancaster comments this is all much more consistent with psychogenic events than with epilepsy. Id.

Videos #409_0328_01 and #409_0329_01, show petitioner's then-husband and Stan Kurtz putting a wet cloth on petitioner's head as she lies on a couch without moving. When asked questions, she shakes her body, but does not answer. Id.

In another video (#409_0337_01), one of Dr. Buttar's employees places an electronic device on petitioner's neck, talking about electric frequencies in every cell of the body. Dr.

Lancaster comments he has never seen anything like this and does not know what this device is supposed to do. Moreover, the process sounded like nonsense to Dr. Lancaster and the testing method described in the video has no scientific or medical basis. Id.

In video #409_0338_01, Dr. Buttar explains the results of this test, saying it shows "lymphatics" and "nutrients." Dr. Lancaster comments he has never heard of a reliable electrical test for either lymphatics or nutrients and could not imagine how it would work. Id.

In four videos (#409_0359_01, #409_0360_01, #409_0360_02, and #409_0360_03), Dr. Buttar states he is very impressed by how effectively petitioner could advocate for the dangers of vaccines "like another Jenny McCarthy." Dr. Buttar sees a connection between petitioner's condition and children with autism, which Dr. Buttar thinks vaccines cause. Dr. Buttar and Stan Kurtz discuss how petitioner could speak for millions of people whom vaccines injured. Id.

In three videos (#409_0361_01, #409_0361_02, and #409_0362_01), Dr. Buttar discusses with petitioner several unusual treatments involving her "interference." Petitioner speaks to Dr. Buttar in a slow, laborious way. She is connected to an IV which appears to remove and return her blood. Id. In video #409_0363_01, Dr. Buttar's staff explains the machine uses ultraviolet light to kill viruses and bacteria in her blood. Dr. Lancaster comments that this treatment has no basis in reliable medicine and does not correspond to any accepted treatment for disease. Moreover, petitioner did not have a bloodstream infection and this method would not treat one. Id.

Videos #409_0368_01 and #409_0369_01 show petitioner talking to Dr. Buttar when she suddenly has jerking of her head when asked to move (something not apparent on a prior video when she made similar movements). Petitioner puts a hand on her chin and stops the jerking. Id.

Videos #409_0380_01 and #409_0381_01 show petitioner in a hyperbaric chamber. Id. at 19. She seems suddenly to have improved speech later in this process. Video #409_0382_01 shows Stan Kurtz saying petitioner spoke very well for three hours after hyperbaric chamber therapy earlier. The video shows petitioner coming out, communicating by gestures. She whispers she can wiggle her toes. She whispers she had a seizure just in her toes. Dr. Buttar plans more therapy with "mist" and "chelators." Id.

Under the subheading "October 20, 2009 videos," video #409_0398_01 shows petitioner preparing to go into the hyperbaric chamber. Id. She has a headshaking episode. Video #409_0401_01 shows petitioner speaking normally in the hyperbaric chamber. Petitioner says she is speaking in her normal voice. She drinks water without difficulty. Id.

Three videos (#409_0402_01, #409_0403_01, and #409_0404_01) show petitioner speaking, moving, and eating in the chamber normally. Her then-husband notes excitedly her remarkable recovery. Id. Video #409_0405_01 shows the hyperbaric chamber being depressurized. Video #409_0406_01 shows petitioner out of the chamber in a wheelchair. Video #409_0407_01 shows Stan Kurtz explaining that petitioner worsened immediately as the chamber depressurized. Id.

Video #409_0430_02 shows petitioner communicating only with gestures in Dr. Buttar's office. Id. Video #409_0430_03 shows petitioner speaking quietly but normally at first, but later unclearly. She is carried from the room. Id.

Three videos (#409_0433_01, #409_0433_02, and #409_0433_03) show petitioner having a video chat with Jenny McCarthy, in which petitioner speaks normally and describes how much she has recovered. Id. at 20. Petitioner says she thought she would die when her tongue paralysis spread to her throat or lungs. She says she could not speak normally because of tongue paralysis. She states she had burning pain in the MRI machine because of mercury in her system. Dr. Buttar then explains his theories on mercury toxicity and his treatments. He states petitioner would not have survived a week without his treatments. He also states the hyperbaric therapy did not use oxygen, just pressure. Petitioner speaks normally with Dr. Buttar; she eats and drinks without difficulty. She appears asymptomatic. Dr. Lancaster comments that petitioner was unaware that mercury is not paramagnetic and therefore the MRI's magnetic field would not have affected her. Id.

In three videos (#409_0434_01, #409_0434_02, and #409_0434_03), petitioner is speaking on the phone with a normal, clear voice. She drinks without difficulty. Her voice and movements are normal. She discusses how important it was to have Fairfax Hospital and Johns Hopkins Hospital to say on camera that flu vaccine caused her symptoms. Id.

Videos #409_0435_01 and #409_0436_01 show petitioner sitting in a chair, speaking normally, while getting an IV. Her movements appear entirely normal. She is very enthusiastic about eating and discusses her immediate recovery with chelation therapy. Id. Video #409_0437_01 discusses how important it is to get the word out correctly about her recovery. Video #409_0438_01 shows petitioner standing and moving very well without evidence of her earlier movement difficulty. Her speech is normal and she seems happy and comfortable. Videos #409_0439_01 and #409_0442_01 show her planning with Dr. Buttar and Stan Kurtz to publicize her story. Id.

Video #409_0446_01 shows petitioner doing various tests of coordination: balancing, touching her finger to her nose, etc. She does very well on these tests. Video #409_0447_01 shows petitioner sticking out her tongue and waving it to demonstrate good tongue coordination. She speaks and walks normally. Id.

Under the heading "October 21, 2009," video #409_0473_01 shows Stan Kurtz saying that petitioner's particular sequence of treatments has never been used before on anyone else. Id.

Videos #409_0493_01 and #409_0494_01 show petitioner speaking to Dr. Buttar and then eating a hamburger, performing both activities without any difficulty. Id. at 21.

Three videos (#409_0514_01, #409_0514_02, and #409_0514_03) show petitioner resting on a sofa apparently in a hotel room or apartment. She speaks in a low, quiet, unclear voice and says her symptoms have returned, which she attributes to mercury. Id. She says her whole body feels hot. Dr. Buttar puts TD-DMPS drops on her arms and asks petitioner to rub

her forearms together. She says she cannot and asks Dr. Buttar to rub her forearms for her. Dr. Buttar says petitioner's nervous system will pick up this treatment. Id.

Video #409_0514_04 shows Dr. Buttar continuing to rub petitioner's forearms together. He says he thinks the treatment will work quickly, and he applies more drops to her forearms. Video #409_0514_05 shows Dr. Buttar saying he thinks petitioner is getting better. She then says, "My tongue is coming back." Her speech soon returns to normal and she remarks, "that was really quick." Dr. Buttar says the cause was mercury. He also mentions viral replication. Petitioner says her lungs are burning and that this will cause a seizure. Petitioner says, "It is moving up here" while pointing to her face, and then says, "Forehead." Her voice is slurred again. Her voice is back to normal with the next sentence and she states again that the feeling is moving, while she speaks in a slow, unclear voice. Within seconds, petitioner is normal again. Id.

Video #409_0514_06 shows Dr. Buttar administering more of the drops to petitioner. In videos #409_0516_01 and #409_0516_02, petitioner and Dr. Buttar discuss how many people have vaccine injuries. In video #409_0517_01, petitioner holds up the bottle of drops to the camera. The bottle says TD-DMPS. Dr. Lancaster comments that there is no sound scientific basis to think a chelator can be applied to the forearms in order to treat mercury toxicity. In addition, a rapid response to these drops emphasizes strongly that petitioner had a psychogenic illness. Id.

Under the subheading "October 22, 2009 videos," video #409_0519_01 shows petitioner eating food and speaking without any difficulty. Video #409_0520_01 shows petitioner placing more of the TD-DMPS drops on her forearms and rubbing them together. Id.

In video #409_0533_01, petitioner is in a car and reports her tongue is numb again. Id.

Video #409_0546_01 shows someone performing a qEEG on petitioner and discussing whether the test showed artifact or seizure. Id. The viewer (i.e., Dr. Lancaster) could see the qEEG partially in the background of the video. The person performing the qEEG states petitioner's headshaking event was a diffuse seizure. Id. Dr. Lancaster found this statement unconvincing because the event looked like muscle artifact that petitioner's headshaking triggered. Id. at 21-22. He said an epilepsy specialist could review the entire data of the qEEG to examine this. Id. at 22. Dr. Ruben Cintron, petitioner's neurologist who is also board certified in neuromuscular electromyography (see supra, n.41) did review this qEEG and found it to be normal (med. recs. Ex. 1, at 14), contrary to the technician Ms. Preston's conclusion when petitioner was under Dr. Buttar's care.

Video #409_0562_01 shows petitioner speaking normally, then pausing, staring off, and having head shaking. Then she speaks in a slow, slurred voice. Dr. Lancaster comments this behavior is most consistent with a psychogenic event. Petitioner returned to her normal voice and then back to her slurred, strained voice a couple of times within a period of minutes. Id.

On November 19, 2009, Fox News had a follow-up showing petitioner walking, laughing, and talking normally after her treatment sessions with Dr. Buttar. Id.

Dr. Lancaster then comments on this case. He notes petitioner developed a complex series of symptoms after receiving flu vaccine on August 23, 2009. She reported a flu-like illness beginning on September 3, 2009. She then went to the ED of a hospital for rhabdomyolysis from which she recovered. Dr. Lancaster defines rhabdomyolysis as a breakdown of muscle fibers with leakage of the muscle enzymes into the blood. There is a risk of kidney damage if the levels of these proteins in the blood are excessive, but petitioner recovered with hydration and supportive care. Dr. Lancaster states rhabdomyolysis is self-limiting and recovery of muscle is generally full. He notes that petitioner manifested full recovery from rhabdomyolysis by running an 8K race. Ex. H, at 22.

Dr. Lancaster says there are many causes of rhabdomyolysis, the most common being substance abuse, exertion, trauma/immobilization/crush injuries, excessive heat, and seizures. Influenza infection may also cause rhabdomyolysis, but only a very few isolated case reports of rhabdomyolysis after flu vaccination exist, often in conjunction with other risk factors such as medications and illness. Id.

Dr. Lancaster notes that before petitioner had rhabdomyolysis, she was regularly engaged in distance running and was training for a half-marathon. He states exertion is a common cause of rhabdomyolysis. Dr. Lancaster's opinion is that it is more likely that factors other than her flu vaccination, i.e., running, a viral infection, or other causes, triggered her rhabdomyolysis. In any event, Dr. Lancaster notes that rhabdomyolysis does not damage the autonomic nervous system or the central nervous system. Rhabdomyolysis would not explain foreign accent syndrome, seizure-like events, tongue paralysis, and other symptoms that petitioner reported. Dr. Lancaster writes he cannot find any logical connection between petitioner's isolated rhabdomyolysis and her subsequent symptoms. Id.

Dr. Lancaster states that from September 17, 2009 to December 2009, multiple physicians treated petitioner for numerous symptoms which had no physiological basis. Id. at 23. These symptoms persisted into other phases of her illness. Multiple physicians suspected petitioner had conversion disorder. Dr. Lancaster gives 11 examples of conversion disorder:

1. On September 17, 2009, petitioner returned to Loudon Hospital, complaining of profound weakness and tingling of all four limbs. She appeared initially to be profoundly weak, but on physical examination, the treating neurologist found no physiological cause for these symptoms. Although she reported having fainted, her orthostatic blood pressure measurements were normal.
2. On September 22, 2009, Dr. Sarfraz A. Chaudhary evaluated petitioner and suspected a psychologic cause. He ordered a lumbar puncture, which was normal.
3. On September 26, 2009, petitioner was admitted to Inova Health System where Dr. Mohammad Mannon suspected a psychiatric etiology for her symptoms.
4. On September 29, 2009, Dr. Paul M. Dellemonache evaluated petitioner for suspected conversion disorder. Dr. Dellemonache noted conversion disorder was a diagnosis of exclusion.
5. On October 2, 2009, Dr. Garry Ho evaluated petitioner for MS. He considered whether petitioner had conversion disorder or another psychiatric etiology.

6. On October 3, 2009, petitioner was admitted to Johns Hopkins Hospital for headaches, pain in her face and neck with speaking, intermittent uncontrollable blinking, difficulty focusing, chills, sweats, lightheadedness, vivid dreams, and difficulty sleeping. Physical examination was notable for elements strongly suggesting she had psychogenic illness, such as "give-way" weakness (which Dr. Lancaster explains is inconsistent effort during strength testing), odd speech patterns, and astasia-abasia (which Dr. Lancaster explains is a wild gait that does not conform to any physiological disorder and requires good balance to perform; it is a sign of conversion disorder). The attending neurologist Dr. Victor C. Urrutia thought petitioner clearly had a strong psychogenic component to her symptoms. He also noted that petitioner's symptoms did not fit a physiological paradigm and that they were related to anxiety after her earlier event of rhabdomyolysis.
7. On October 6, 2009 and October 13, 2009, Dr. Garry Ho again considered diagnosing petitioner with conversion disorder.
8. On December 7, 2009, Dr. Randolph R. Stevenson, a neurologist, noted petitioner had a very clear astasia-abasia when walking. Her tremor was highly distractible and strongly suggestible. Dr. Stevenson opined petitioner had a very clear functional component to the majority of her examination.
9. On March 16, 2010, Dr. Ruben Cintron, a neurologist who had seen petitioner multiple times, thought her symptoms were still bizarre neurologically. Petitioner had an accent which fluctuated. She was able to walk sideways but not forward. But there was no objective evidence on examination of etiology.
10. On January 10, 2012, Dr. Patrick Lyden, a neurologist, evaluated petitioner and thought she had an unsuspected psychiatric diagnosis, but it was too soon to be sure.
11. On January 13, 2012, Dr. Lyden saw petitioner again and noted the lack of evidence that petitioner had ever had GBS. He also noted the lack of evidence that petitioner had hypotension. He described her tilt-table test result as normal although she had very unusual symptoms during the test. She had a dystonic and syncopal attack during a carotid ultrasound. During physical examination, petitioner had a foreign accent for half the examination. Dr. Lyden opined that a number of petitioner's symptoms were embellished. Dr. Lyden thought a good psychologist should see petitioner and referred her to Dr. Basian.

Id. at 23-24.

Dr. Lancaster defines conversion disorder as a psychiatric illness in which symptoms such as numbness or weakness appear to be neurologic or medical, but they lack a physiological basis. The cause of these symptoms is a subconscious reaction to stress. Dr. Lancaster distinguishes conversion disorder from malingering. Patients with conversion disorder are not consciously aware of what they are manifesting and thus do not voluntarily control it. Common manifestations of conversion disorder are: psychogenic seizure-like events; bizarre gait; reported numbness or weakness; events resembling syncope; difficulty swallowing; and tremor. Dr. Lancaster states persons with conversion disorder may be highly suggestible and stress may provoke their symptoms. He notes that conversion disorder is common in neurologic clinics. Up

to 14 percent of patients seeking neurological care do not appear to have a physiological cause of their symptoms and may have conversion disorder. Particularly common are psychogenic seizure-like events and a common diagnosis during evaluation of patients who appear to have epilepsy. Because of the lack of specific diagnostic tests, doctors are cautious about diagnosing conversion disorder. However, Dr. Lancaster notes that there is a low frequency of patients initially diagnosed with conversion disorder who have a subsequent medical explanation for their symptoms. Id.

Dr. Lancaster opines that the symptoms petitioner displayed between September and October 2009, as the medical records describe and the videos display, support the diagnosis of conversion disorder. These symptoms include: periods of speaking with a foreign accent; unexplained weakness and numbness of her limbs; reported paralysis of her tongue; an unusual (non-physiological) jerking gait while maintaining her balance; memory complaints; the ability to walk backward, but not forward; and the ability to run forward but not to walk forward. These symptoms were inconsistent during the time period of the video displays. For example, Dr. Lancaster notes that petitioner reported paralysis of her tongue but then she ate and spoke normally at times. She reported profound cognitive and reading problems, but then would send e-mail on her computer. She reported profound weakness, but then ran an 8K race. She reported profound unsteadiness, but never fell regardless of her wild gyrations. Id.

Dr. Lancaster notes that petitioner underwent MRI, EMG, and lumbar puncture tests, but they did not show any physiological basis for her symptoms. Dr. Lancaster states it is difficult to imagine a disorder that would cause all of petitioner's symptoms without any objective findings on any of the tests of petitioner's central and peripheral nervous systems. He recalls that multiple skilled neurologists thought petitioner's symptoms were non-physiological. In addition, the varying severity of the symptoms and the ease and frequency with which they disappeared and reappeared with sensory tricks and other non-traditional treatment suggest a psychogenic etiology. Although conversion disorder is a diagnosis of exclusion, Dr. Lancaster notes that thorough diagnostic testing, including repeated examinations, brain MRIs, and lumbar puncture, excluded any other plausible cause. He concludes that the most logical explanation of petitioner's events in this time frame is conversion disorder. Id.

He notes that the events in Dr. Rashid Buttar's office, beginning on October 19, 2009, strongly support a diagnosis of conversion disorder. Id. In this visit and subsequent ones, petitioner appeared unable to speak, to have profound cognitive problems and seizure-like episodes, and to be profoundly weak. Id. at 24-25. Dr. Buttar diagnosed petitioner with mercury toxicity from her flu vaccination. Id. at 25. At other times, Dr. Buttar mentioned infections, autoimmunity, and other theories as causative. Dr. Lancaster opines these diagnoses are implausible. Dr. Buttar administered a series of alternative treatments, including EDTA (ethylenediaminetetraacetic acid), DMPS (dimercaptopropanesulfonic acid), hyperbaric chamber, and some sort of nerve stimulation. Id. The videos show petitioner responding dramatically to multiple different treatments, including the hyperbaric chamber without oxygen, the chelators, and the TD-DMPS drops that Dr. Buttar placed on her forearms. Dr. Lancaster says the most likely explanation of these events is conversion disorder with petitioner's strong

response to the suggestion that she would get better with these treatments. For example, petitioner responded within seconds to minutes of Dr. Buttar's applying drops to her forearms with a dramatic recovery of her speech. Dr. Lancaster says it is difficult to imagine any biological basis for her response. He says the most likely explanation is conversion disorder responding to suggestion. Id.

Dr. Lancaster continues by noting subsequent reports, including extensive video and audio of petitioner on "20/20" (the ABC interview show) and in the videos Dr. Lancaster already described in his expert report show an apparently psychogenic gait disorder with features of astasia-abasia. Id. Petitioner would lean forward and lurch wildly while flailing her arms. Dr. Lancaster notes that this gait requires someone with very good balance to perform it without falling and is not consistent with dystonia or any other neurological disorder in Dr. Lancaster's experience. He says petitioner's foreign accent syndrome and trouble speaking which she exhibited during the "20/20" show also seems psychogenic. Dr. Lancaster points out that foreign accent syndrome has rarely been reported after structural brain injuries, but petitioner has never had a structural brain injury. Therefore, psychogenic etiology is most likely. Moreover, the quality of petitioner's speech problem varies over time. Her voice ranges from being strained, slurred, staccato, and quiet, to a clear British-sounding accent. Id.

Dr. Lancaster emphasizes that it is important to note that petitioner's main symptoms (gait, speech, psychogenic seizures, tongue paralysis causing trouble eating) during this period of time have nothing to do with the autonomic nervous system. Id. Petitioner's ability to run an 8K race while her symptoms resolved during running demonstrates she has excellent cardiovascular autonomic function. Moreover, her cardiology work-up was normal. Petitioner's eating difficulties reportedly involved tongue movements, not the autonomic aspects of digestion. Dr. Lancaster notes the dramatic fluctuation of petitioner's gait symptoms, gait nature, and ability to run but inability to walk forward argue strongly for a psychogenic gait disorder.

Similarly, petitioner's speech problem is most consistent with a psychogenic disorder. Dr. Lancaster says the sudden resolution of petitioner's speaking problem as soon as she starts to run is inconsistent with any neurological disorder and certainly not a neurological disorder with an autoimmune etiology. Dr. Lancaster's opinion is that the seizure-like events were psychogenic non-epileptic seizures. They involved generalized tremulousness and apparent weakness, but never a fall or convulsion. Petitioner retained the ability to respond during many of these events. Dr. Lancaster says that her events responded dramatically to treatments that do not affect epilepsy, such as chelation and hyperbaric chamber without oxygen. Id. Dr. Lancaster says the video evidence strongly argues against petitioner having autonomic dysfunction at this time and shows diverse manifestations of conversion disorder. People around her who focused on her symptoms and proposed various theories for them influenced petitioner's reported symptoms. For instance, after hearing about mercury coming from her body, petitioner reported feeling mercury coming out and affecting in a migratory fashion her chest, mouth, and head. Id.

Dr. Lancaster recounts that by December 2009, many doctors were evaluating petitioner for possible autoimmune dysfunction. Ex. H, at 26. He notes the following evaluations from various doctors from December 2009 to April 2012:

1. On December 18, 2009, Dr. Mark Tanenbaum, a cardiologist, found petitioner did not have cardiac abnormalities. A stress cardiogram showed normal heart rate and blood pressure responses to exercise, normal exercise capacity, no arrhythmia, and transient syncope post-exercise secondary to neurocardiogenic hypotension. Dr. Lancaster says it is unclear whether this represented true or psychogenic syncope. He thinks in the overall context of petitioner's case, a psychogenic cause is more likely.
2. The results of a brain MRI and a brain PET/CT scan were normal.
3. On June 4, 2010, Dr. Zangeneh, an endocrinologist, concluded petitioner's adrenal, thyroid, and other endocrine systems were normal.
4. On July 15, 2010, petitioner underwent a tilt-table test. She was tilted upright and developed slurred speech progressing to a total inability to speak over 15 minutes. However, blood pressure and heart rate were normal during the entire time. Petitioner was laid flat and her dystonia resolved immediately, but her speech remained slurred for nearly one hour. The report concluded that petitioner had a normal heart rate and blood pressure response to tilt-table testing. Dr. Lancaster says this study is strong evidence that petitioner's speech and language symptoms had nothing to do with blood pressure changes or autonomic function at all. He considers this description of her symptoms is remarkably similar to her speech problems recorded on various videos. Her autonomic function was totally normal, but her symptoms were profound.
5. On August 4, 2010 and August 25, 2010, Dr. Frisca Yan-Go, whom Dr. Lancaster describes as a very well-respected autonomic specialist, evaluated petitioner and concluded petitioner has a very complex symptomatology, many symptoms of which Dr. Yan-Go could not explain by a unified disorder. Dr. Yan-Go stated she was worried about whether petitioner had some functional overlay and subconscious effect of conversion reaction.
6. On August 30, 2010, Dr. Kevin Ghassemi, a gastroenterologist at UCLA, evaluated petitioner. She had a test called esophageal manometry and an upper GI series, the results of which were normal.
7. On September 9, 2010, petitioner had a repeat tilt-table test. Petitioner's blood pressure did not drop, but there was one data point where she had elevated pulse. Petitioner's response to deep breathing was normal.
8. On September 14, 2010, Dr. Frisca Yan-Go noted petitioner's GI evaluation did not show any true motility problem, and concluded that, overall, this did not point to a serious pure autonomic failure or neurodegenerative dysautonomia.
9. On October 15, 2010, Northwest GI associates measured petitioner's blood pressure and pulse over nine times after she ate a meal. The results were within the normal range.
10. On December 15, 2010, Dr. Daniel Wilkson, a cardiologist, reviewed petitioner's Holter monitor study, which did not show evidence of arrhythmia. Petitioner did not have any pauses or abnormal heart beat to explain her symptoms.
11. On August 12, 2011, a brain SPECT scan showed severe bilateral watershed perfusion deficits, which the technician said supported a diagnosis consistent with

lupus or vasculitis, even though, as Dr. Lancaster points out, the medical records do not support a diagnosis of lupus or vasculitis. Dr. Lancaster thinks this SPECT scan conclusion was a false positive. He notes that petitioner's subsequent normal brain PET scan suggests the SPECT scan interpretation was incorrect.

12. On March 30, 2012, petitioner underwent a gastric emptying study, which showed markedly prolonged gastric emptying. Id. Dr. Lancaster notes that petitioner's medications around this time (April 18, 2012) included Sandostatin, metoclopramide, gabapentin, disopyramide, midodrine, fludrocortisone, Depo-Provera, acyclovir, cerefolin, and methylprednisolone dose pack (apparently on August 31, 2011), some of which could affect gastric motility.
13. On April 18, 2012, Dr. Daniel Wallace, a rheumatologist, evaluated petitioner and reported she felt better after she received IV steroids that Dr. Olek prescribed, but did not mention that she improved objectively.

Id. at 26-27.

Dr. Lancaster concludes from this summary that preponderant evidence suggests petitioner's autonomic function was intact. Id. Multiple objective measures of petitioner's cardiovascular responses (tilt-table testing) and autonomic GI function (GI series) were objectively normal. Dr. Yan-Go, whom Dr. Lancaster describes as a specialist in the autonomic nervous system, did a thorough evaluation of petitioner and did not find evidence of significant autonomic dysfunction. Dr. Wilkinson, a cardiologist, conducted extensive cardiac monitoring of petitioner to detect abnormal heart rhythms and did not found any abnormality. Dr. Ghassemi, a gastroenterologist, did a GI series to evaluate petitioner for failure of autonomic motility of her GI system and did not find any abnormalities. Northwest GI associates tried to document a post-prandial, i.e., after eating, drop in blood pressure but instead found petitioner had multiple normal pulse and blood pressure readings over one hour after she ate a meal. The one abnormal test result Dr. Lancaster found (the September 9, 2010 tilt-table test) reported a rise in one pulse measurement after tilting in the context of multiple normal pulse measurements and maintained blood pressure. Dr. Lancaster notes that petitioner's response to deep breathing, i.e., another autonomic cardiovascular measurement, was normal. Petitioner's medical records did not document any problem petitioner had with pupillary reactivity or sweating, which are also autonomic functions.

Dr. Lancaster sums up that looking at petitioner's clinical picture and diagnostic evaluations in toto, he does not think the isolated data point on the September 9, 2010 tilt-table test provides sufficient evidence that petitioner had any autonomic disorder through the end of 2010. Ex. H, at 27. He notes that petitioner's profound symptoms on tilt-table testing, including dystonic posturing and inability to speak normally for almost an hour, occurred even though she had normal blood pressure and heart rate. He says autonomic failure would cause neurological symptoms during a tilt-table test only if the test prevented the maintenance of normal blood pressure, resulting in decreased perfusion of the brain and syncope (passing out) or presyncope (feeling lightheaded). These symptoms would resolve very quickly, taking seconds to one to two minutes when the patient returns to the supine position. If petitioner failed to maintain adequate

blood pressure, her mentation might change due to an autonomic cause, but this change would not occur during normal pressure. He concludes therefore that dysautonomia would not explain petitioner's symptoms during her tilt-table test.

Dr. Lancaster also explains that petitioner's rapid expulsion of food from her mouth during the GI studies is not consistent with autonomic failure. Her earlier studies showed she has normal GI motility throughout her enteral system once she swallowed food. Autonomic failure could produce vomiting from a lack of forward motility in the esophagus, stomach, and intestines. But initiation of swallowing is under voluntary control, not under autonomic control. Therefore, petitioner's expulsion of food from her mouth was not due to autonomic failure during the GI study. Id.

After Dr. Lancaster watched hours of petitioner's earlier speech and eating symptoms on the videos, he concludes that the most likely cause of her symptoms was of the same nature as her earlier conversion disorder symptoms, i.e., the cause for these symptoms was psychogenic, not autonomic. Id. He notes that a later GI study on March 30, 2012 showed delayed gastric emptying, but this result conflicts with the results of prior testing, which he attributes to a false positive. Id. at 28. But, even if it did prove autonomic GI failure, it occurred many months after petitioner's August 23, 2009 flu vaccination and, moreover, petitioner was taking medications that could have affected her GI motility by March 30, 2012, thus influencing the results of that GI study.

Dr. Lancaster states the results of the brain SPECT scan were not diagnostic of a particular illness. Petitioner's results on other brain imaging, i.e., PET/CT of the brain, brain MRI, suggest the results of her SPECT scan were probably a false positive. Dr. Lancaster notes that if petitioner did have central nervous system vasculitis, he would have expected her to have a series of cerebral infarctions that would have caused different symptoms and revealed objective abnormalities on her brain MRI.

Dr. Lancaster believes Mission Hospital had insufficient evidence to diagnose petitioner with myasthenia gravis. She tested negative for two antibodies used to diagnose myasthenia gravis (AChR and MuSK). He notes that 85 percent of myasthenia gravis patients have AChR antibodies and an additional 5 percent have MuSK antibodies. Dr. Lancaster emphasizes that at least one of these antibodies is present in the great majority of patients with myasthenia gravis. To diagnose the remaining seronegative myasthenia gravis patients, electrodiagnostic studies such as repetitive nerve stimulation and single fiber EMG can support the diagnosis. (At the time of Dr. Lancaster's first expert report, petitioner had not taken these tests. But when she did later on, the results were normal.) Dr. Lancaster concludes that if all these tests are negative, systemic myasthenia gravis is very unlikely to be the correct diagnosis. Given petitioner's previously unexplained neurologic symptoms, which he attributes to conversion disorder, Dr. Lancaster thinks petitioner's later symptoms suggesting myasthenia gravis are also due to conversion disorder. He concludes the evidence in the exhibits is insufficient to diagnose petitioner with myasthenia gravis.

Dr. Lancaster states the autonomic nervous system controls multiple bodily systems, most of them outside complete conscious control or awareness. Id. The primary systems that the autonomic nervous system controls, and the consequences of autonomic failure, manifest in: (1) control of blood pressure/orthostatic hypotension; (2) sweating/anhidrosis; (3) bladder function/urinary retention; (4) sexual function/sexual dysfunction; and (5) gastrointestinal motility/dysphagia, regurgitation, bloating, and pain.

Dr. Lancaster notes that the autonomic nervous system does not control consciousness, memory, speech, movement, sensation, or gait. A patient who has autonomic failure may have syncope when assuming an upright posture, but he or she should not have problems with cognition. A patient may fall from a decrease in blood pressure, but his or her gait should otherwise be normal. Dr. Lancaster says dysautonomia cannot cause a flailing or wild gait and cause a difference in the ability to walk forward and backward. He also says that the ability to run but not to walk forward is inconsistent with autonomic failure. If a person can maintain blood pressure while running, he or she should be able to maintain it while walking. He notes running makes greater demands on the autonomic blood pressure control mechanisms than walking does. Id.

Dr. Lancaster lists many potential causes of autonomic disease: (1) neurodegenerative conditions, e.g., Parkinson's or multisystem atrophy; (2) peripheral neuropathies, e.g., GBS and many others; (3) Sjögren's syndrome; (4) paraneoplastic disorders; and (5) autoimmune ganglionopathies. Id. He states while many types of peripheral neuropathy could affect the autonomic nerves, GBS is an autoimmune neuropathy that typically causes severe neuropathy with autonomic dysfunction over a period of days to weeks. Id. at 29. Patients with GBS do not get autonomic involvement without involvement of non-autonomic nerves. In a study Dr. Lancaster provided for respondent to file, the primary autonomic finding was tachycardia; no GBS patient had orthostatic hypotension. Another study of GBS patients had autonomic symptoms of bradycardia and hypertension, but not GI symptoms. In all the cases, the autonomic symptoms resolved within six months. Dr. Lancaster states, even though petitioner did not have GBS, and had problems completely inconsistent with GBS (foreign accent syndrome, speech problems), the autonomic symptoms she reported are not of the type or duration of autonomic symptoms seen in GBS patients with GBS-related autonomic dysfunction.

Dr. Lancaster states that autoimmune autonomic neuropathy, also known as autoimmune autonomic ganglionopathy ("AAG"), is an autoimmune disorder in which the immune system targets autonomic ganglia neurons. Id. Unlike GBS, AAG primarily affects only the autonomic nervous system. Some patients have a receptor on autonomic ganglia called the ganglionic acetylcholine receptor. AAG patients have severe problems with autonomic function, including: abnormal lack of pupillary reactivity; fixed heart rate; orthostatic hypotension; anhidrosis; dry mouth/eyes; sexual dysfunction; urinary dysfunction; and GI dysmotility. When the autoantibodies are present together with appropriate symptoms, a doctor can make the diagnosis of AAG confidently. When someone does not have a measurable antibody presence, the diagnosis of AAG is less likely. Dr. Lancaster says petitioner had no evidence of AAG despite multiple careful evaluations of pupillary abnormalities, dry mouth/eyes, urinary dysfunction,

sexual dysfunction, fixed heart rate, or anhidrosis. Her test results of orthostatic hypotension were normal or unconvincing. Her GI evaluations had mixed results, but the studies done within a reasonable proximity to her August 23, 2009 flu vaccination were normal. Dr. Lancaster regards petitioner's diagnosis of AAG as unlikely for three reasons: (1) her autonomic symptoms and findings were atypical for AAG; (2) she had many non-autonomic symptoms; and (3) the only specific diagnostic test for antibodies was negative. Id.

Dr. Lancaster examines the results of studies of Fluzone vaccine and dysautonomia and finds a paucity of evidence linking autoimmune autonomic neuropathy with vaccines of any kind. Id.

As for temporal interval between the flu vaccination and petitioner's onset of symptoms, Dr. Lancaster notes petitioner had an episode of rhabdomyolysis 20 days after vaccination and 9 days after a flu-like illness. Id. at 30. He comments that there are case reports of flu vaccination followed by rhabdomyolysis, but he considers these case reports insufficient to establish causation. Dr. Lancaster states that exertion is a more common cause of rhabdomyolysis and a more likely explanation of petitioner's symptoms. He considers it unlikely that petitioner's rhabdomyolysis was related to her flu vaccination. He notes petitioner recovered from rhabdomyolysis rapidly and it did not cause her persistent disability. He also notes that her rhabdomyolysis did not have a logical connection to petitioner's subsequent symptoms which petitioner's expert Dr. Steinman attributes to autoimmune dysautonomia. Id.

Continuing with his analysis of timing, Dr. Lancaster says petitioner's neurological symptoms of numbness, weakness, shaking, cognitive complaints, tongue paralysis, and foreign accent syndrome began 25 days after she received flu vaccine. He admits that this is a plausible time frame for a vaccine-induced illness. However, he states no neurological disorder including autonomic nervous system disorder explains petitioner's symptoms after extensive work ups. He attributes her symptoms to conversion disorder. Id.

Dr. Lancaster notes that petitioner's doctors did not focus on autonomic symptoms until 2010, which was over three months from her August 23, 2009 flu vaccination. In contrast, from September to December 2009, petitioner's primary complaints were speech disruption, reported weakness, and seizure-like events, which were indicative of central nervous system, not autonomic nervous system, symptoms. Id. Throughout 2010, the opinion of autonomic experts such as Dr. Yan-Go was petitioner did not have any significant autonomic dysfunction. The first apparently abnormal GI study was in 2012, over two years after petitioner's flu vaccination. He says that in order to link any autonomic symptoms to petitioner's August 23, 2009 flu vaccination, the symptoms would have to be part of a single syndrome with neurologic symptoms. Dr. Lancaster states petitioner's theory of the case fails to explain the neurologic symptoms and focuses just on the later autonomic symptoms. He says her case therefore involves too long a time frame between the vaccination and her autonomic symptoms. Id.

Dr. Lancaster finds several logical gaps in petitioner's proposed chain of causation of alleged serious autoimmune dysautonomia, a type of inflammatory autoimmune neuropathy, after flu vaccination, as Dr. Steinman proposed in Exhibit 65. First, Dr. Lancaster says

autonomic failure does not explain many of petitioner's most prominent symptoms. He says Dr. Steinman's diagnosis is therefore incorrect. Secondly, Dr. Lancaster states there is no evidence that petitioner had autonomic failure in temporal proximity to her flu vaccination. Thirdly, Dr. Lancaster says petitioner's clinical history and presentation do not fit the diagnosis of autoimmune disorders with prominent autonomic symptoms, such as AAG and GBS. Fourthly, no evidence links Fluzone vaccine to autonomic failure or AAG. Id.

Dr. Lancaster states that conversion disorder, a non-vaccine related alternate cause of petitioner's most prominent symptoms, explains her lurching gait, abnormal movements, spells of decreased responsiveness, reports of numbness/weakness, foreign accent syndrome, and periods of speech arrest. Moreover, he says conversion disorder is entirely consistent with the diverse and fluctuating nature of her symptoms. Id. He continues that conversion disorder explains petitioner's response to suggestion and Dr. Buttar's multiple treatments (hyperbaric chamber, drops on her forearms, chelation). Id. at 30-31. Dr. Lancaster states he cannot conceive of a medical illness responding so dramatically to any of these treatments, let alone to all of them. Id. at 31. Moreover, Dr. Lancaster says the negative findings on MRI studies, lumbar puncture, nerve conduction studies, and many autonomic tests are entirely consistent with the diagnosis of conversion disorder. He states, "The only logical diagnosis that could produce such diverse findings over such a long period of time without leaving objective abnormalities on these tests is conversion disorder." Id.

Dr. Lancaster notes that many of petitioner's doctors suspected conversion disorder or another psychiatric cause of petitioner's symptoms. While some of her doctors agreed with the diagnosis of autonomic failure, others conducted careful evaluations and did not find any evidence to support the diagnosis of autonomic failure. Dr. Lancaster as well does not see evidence that petitioner had autonomic failure in the months following her flu vaccination. Id. He says, in reviewing all of petitioner's medical records and the videos filed in the case, he is struck with how the great majority of petitioner's symptoms had nothing to do with the autonomic nervous system. He says that doctors who enunciate a theory of petitioner's case focusing on the autonomic nervous system without accounting for petitioner's symptoms such as the ability to walk backward or sideways but not forward, unusual jerking movements while walking, ability to run better than walk, cognitive difficulties, language arrest, and speaking with a foreign accent have "fundamentally failed to explain the actual cause" of petitioner's alleged disability. Id.

Dr. Lancaster then comments on Dr. Steinman's expert report (Ex. 65). He says Dr. Steinman's theory is that petitioner developed "serious autoimmune dysautonomia, a type of inflammatory neuropathy," while dismissing the opinions of doctors at Johns Hopkins Hospital that petitioner had conversion disorder. Dr. Lancaster thinks the Johns Hopkins doctors are correct. Multiple treating doctors at different times and in different facilities suspected or diagnosed petitioner with conversion disorder. Their findings included inconsistencies in petitioner's neurological examination (astasia-abasia, speaking with a British accent, unexplained weakness/numbness, speech arrest) which Dr. Steinman does not explain. Dr.

Lancaster viewed the extensive symptoms in hours of videos and could arrive at no other explanation for them except conversion disorder. Ex. H, at 31.

Dr. Lancaster says Dr. Steinman relied on doctors' opinions that were not convincing proof of autonomic dysfunction in order to diagnose autonomic dysfunction himself. Id. In many of these medical records, Dr. Lancaster states the treaters relied on petitioner's giving them her diagnosis without making that diagnosis themselves. Objective testing of petitioner was mainly negative and Dr. Yan-Go, whom Dr. Lancaster described as the most expert practitioner of autonomic neuropathy, decided petitioner did not have significant autonomic dysfunction based on objective findings.

Dr. Lancaster's biggest problem with Dr. Steinman's opinion is that Dr. Steinman fails to address petitioner's most serious and disabling symptoms: profound language disturbance including foreign accent syndrome and periods of inability to speak or walk forward (while maintaining the ability to walk backward and respond to sensory tricks), ataxia, tremors, and cognitive complaints. Id. Dr. Lancaster says Dr. Steinman "simply qualifies these symptoms as 'relevant' to his diagnosis of autoimmune dysautonomia (Ex. 93)." Id. at 31-32. Dr. Lancaster notes that petitioner's symptoms do not pertain to the autonomic nervous system, but instead to the central nervous system. Id. at 32. He reiterates that Dr. Steinman's diagnosis based on autonomic dysfunction ignores the most salient clinical features and does not support Dr. Steinman's causation theory because it is not a reliable explanation for petitioner's symptoms.

Dr. Lancaster takes exception to Dr. Steinman's thesis that flu virus, containing a small peptide fragment similar to a peripheral nerve protein (myelin basic protein) can produce an autoimmune response to peripheral nerve myelin, causing a demyelinating neuropathy. Dr. Lancaster has three problems with Dr. Steinman's thesis:

(1) Why would this cause only an autonomic neuropathy when most myelin basic protein is located in other parts of the nervous system? Myelin basic protein is particularly prevalent in peripheral nerve and brain. If Dr. Steinman's proposed mechanism were true, petitioner would have developed GBS with perhaps inflammation of the brain and spinal cord. But she did not have GBS.
(2) The medical records do not show that petitioner has antibodies to myelin basic protein.
(3) People have widely studied autoimmunity to myelin basic protein in the context of MS, an autoimmune disease targeting myelin in the central nervous system. Dr. Lancaster says MS does not affect the peripheral autonomic nervous system. Moreover, petitioner does not have MS. Her brain MRI and lumbar puncture were normal. Dr. Lancaster finds Dr. Steinman's theory a poor explanation for petitioner developing autonomic symptoms since the explanation requires hypothetical antibodies (antibodies which no doctor ever measured) to do something very different from what doctors would expect them to do and to cause a disease with which doctors have never associated them.

Id.

Dr. Lancaster notes that Dr. Steinman mentions autonomic dysfunction occurs in many GBS patients. Dr. Lancaster says it is extremely important to recognize that autonomic dysfunction occurs in GBS when there is widespread demyelination of other, non-autonomic nerves resulting in objective weakness, measurable abnormalities on nerve conduction studies, dropped reflexes, and elevated CSF protein. Dr. Lancaster states petitioner never had any GBS findings. Although some of her doctors pondered whether she had GBS, petitioner's preserved reflexes, normal EMG/NCS, and normal CSF protein ruled out the diagnosis of GBS. Id.

Dr. Lancaster comments on Dr. Steinman's analysis of autoimmune autonomic neuropathy ("AAN")/ganglionopathy ("AAG"). Dr. Lancaster notes a doctor can confirm a patient has AAG with a specific autoantibody test, but no one did that test on petitioner. AAG involves generally persistent, severe, and objectively demonstrated failure of multiple components of the autonomic nervous system. However, petitioner lacked features of AAG, such as fixed pupils. Moreover, AAG would not explain foreign language syndrome, gait problems, ataxia, cognitive disability, and many other symptoms petitioner had which do not pertain to the autonomic nervous system. Id.

Dr. Lancaster notes the absence of convincing evidence showing that petitioner's symptoms responded to immune therapies. Her symptoms responded very rapidly to Dr. Buttar's diverse alternate treatments, but Dr. Lancaster views it as implausible for an autoimmune disorder to respond so rapidly to any therapy or to treatments such as chelation or hyperbaric chamber. The evidence does not persuade him that petitioner had autoimmune autonomic neuropathy or any related unspecified disorder. Id.

Dr. Lancaster takes issue with Dr. Steinman's linking autoimmune autonomic ganglionopathy to flu vaccine. The four studies Dr. Steinman cites do not support his thesis. Id. One paper relies on an earlier paper which itself does not cite a reference or facts about a specific case. Id. at 33. The third paper does not discuss vaccines. The fourth paper discusses animal models but no evidence that AAN occurs in people after flu vaccination or any other vaccination. Dr. Lancaster states that the specific antigen in AAN is a form of the acetylcholine receptor present in autonomic ganglia. Dr. Lancaster says if Dr. Steinman's diagnosis of AAN were correct, this is problematic for his theory of molecular mimicry. The problem is what component of flu vaccine mimics or resembles the ganglionic acetylcholine receptor. Dr. Lancaster asks if there is some other antigen on autonomic ganglia, what is that antigen and how closely does a flu vaccine component resemble it? Moreover, where is the evidence that any of this is true? Id.

Dr. Lancaster takes further issue with Dr. Steinman's first supplemental report (Ex. 92) in which Dr. Steinman says only 50 percent of patients with presumed autoimmune autonomic neuropathy have antibodies to the ganglionic acetylcholine receptor. Patients who have those antibodies have important proof of the autoimmune nature of the disease. Dr. Lancaster cites an article,[129] in which the authors at the Mayo Clinic propose criteria for diagnosing autoimmune gastric dysmotility: subacute autonomic GI symptoms, a personal or family history of

[129] Eoin P. Flanagan et al., Immunotherapy trial as diagnostic test in evaluating patients with presumed autoimmune gastrointestinal dysmotility, 26 NEUROGASTROENTEROL MOTIL 1285 (2014). This was filed as Ex. V.

autoimmunity, clear objective proof of autonomic GI dysfunction that objectively improves with immune therapy. Id. Dr. Lancaster states petitioner did not satisfy these criteria. Id.

Dr. Lancaster notes that Dr. Steinman thinks the diagnosis of GBS is pertinent in this case even though petitioner did not have GBS (intact reflexes, normal CSF protein, normal strength). He says Dr. Steinmann seems to imply that petitioner had a purely autonomic form of GBS, although there is no objective evidence to support this diagnosis. The doctors who discussed whether or not petitioner had GBS were not neurologists. Petitioner's neurologists rejected the diagnosis of GBS. Id. Dr. Steinman does not address petitioner's many reported symptoms (dramatic seizure-like episodes, foreign language syndrome, unusual movement problems such as only being able to walk backward or sideways) which no dysfunction of the autonomic nervous system can explain. Id. 33-34.

Dr. Lancaster says very specific objective diagnostic findings exist to diagnose myasthenia gravis, such as specific antibody tests (AChR, MuSK) and electrodiagnostic findings (repetitive nerve stimulation, single fiber EMG). Ex. H, at 34. He notes that petitioner did not have either of the antibody markers. The records do not support electrophysiological evidence of myasthenia gravis. In the absence of this evidence, Dr. Lancaster thinks petitioner's having myasthenia gravis is unlikely. If she developed myasthenia gravis years after her 2009 flu vaccination, there was no relationship between the vaccination and her myasthenia gravis. Id.

Referring to Dr. Steinman's second supplemental expert report (Ex. 108), Dr. Lancaster notes that Dr. Steinman mentions the autonomic manifestations of GBS without mentioning they occur in the context of GBS in the non-autonomic nervous system, i.e., lessened reflexes, weakness, numbness, abnormal nerve conduction studies, elevated CSF protein, none of which petitioner had. Id. Dr. Steinman notes that autonomic symptoms of GBS rarely persist, which to Dr. Lancaster means Dr. Steinman is invoking a purely autonomic form of GBS for which no reliable diagnostic criteria exist. Dr. Lancaster also says Dr. Steinman's analysis does not explain the atypical features petitioner manifested that GBS never causes: dystonia, foreign accent syndrome, speech arrest, etc. Dr. Lancaster finds Dr. Steinman's analysis has too many novel, atypical, or inconsistent elements to be acceptable or medically reliable. Id.

Referring to Dr. Steinman's chart entitled "Analysis of Doctors Who Agree Autoimmune Dysautonomia" (Ex. 93), Dr. Lancaster notes that some of the treating doctors Dr. Steinman cites clearly do not agree with the diagnosis of autonomic failure. Id. Dr. Lancaster also says that while other treating doctors mention the diagnosis of autonomic failure, they do not support it with facts. Dr. Lancaster therefore disagrees with several parts of Dr. Steinman's chart that constitutes Ex. 93:

1. Dr. Lancaster states Dr. Yan-Go did not agree with the diagnosis and thought petitioner did not have dysautonomia. Dr. Yan-Go suspected petitioner had conversion disorder. On August 25, 2010, Dr. Yan-Go, who is affiliated with UCLA, evaluated petitioner and noted petitioner had a very complex symptomatology, much of which Dr. Yan-Go could not explain by a unified disorder. Dr. Yan-Go also wrote she was worried about whether petitioner had some functional overlay and a

conversion reaction. On September 14, 2010, Dr. Yan-Go wrote she still thought overall that petitioner did not have a serious, pure autonomic failure or neurodegenerative dysautonomia.

2. Dr. Lancaster describes Dr. Buttar's diagnosis as mercury toxicity from a vaccine. He notes Dr. Buttar did not focus on autonomic symptoms. The many hours of video of petitioner in Dr. Buttar's office did not involve autonomic nervous system symptoms. In addition, Dr. Buttar's treatments were inconsistent with Dr. Steinman's theory of causation.
3. Dr. Lancaster notes Dr. Atiga did not address and may not have been aware of petitioner's extensive non-autonomic symptoms early in her disease course. Dr. Atiga suspected autonomic dysfunction but did not produce support for that diagnosis. Dr. Atiga referred petitioner to Dr. Yan-Go for an evaluation of whether petitioner had autonomic failure.
4. Dr. Lancaster says Dr. Cintron occasionally suspected an autonomic disorder, but never proved petitioner had one. Dr. Cintron noted the bizarre nature of petitioner's symptoms and his uncertainty about her diagnosis particularly in light of the lack of objective evidence on physical examination about what petitioner's problem was. Dr. Cintron noted her bizarre symptoms of fluctuating accent and ability to walk sideways but not forward. Later in his course of analysis, Dr. Cintron proposed focal dysregulation of cerebral blood flow to explain petitioner's case. Dr. Lancaster says he could not find any precedent for focal dysregulation of blood flow causing someone to walk backward but not forward, to speak in a foreign accent, to have seizure-like events, or petitioner's other neurological symptoms. Dr. Lancaster regards Dr. Cintron's diagnosis of focal dysregulation of cerebral blood flow as incorrect.
5. Dr. Lancaster states Dr. Ghassemi thought autonomic causes of petitioner's GI symptoms were unlikely. On August 26, 2010, given petitioner's many non-GI symptoms, Dr. Ghassemi ordered a test for GI motility. On August 30, 2010, petitioner underwent a gastroesophageal manometry and an upper GI series, both of which were normal. Dr. Lancaster says the normal results of these tests confirmed Dr. Ghassemi's earlier suspicion that petitioner's symptoms were not autonomic.
6. Dr. Lancaster notes that Dr. Ho's diagnosis of dysarthria and stuttering does not support a diagnosis of autonomic failure and these symptoms do not result from autonomic failure. Although Dr. Steinman cites Dr. Ho's notation of dystonia as supportive of Dr. Steinman's diagnostic theory, Dr. Lancaster notes that Dr. Ho recorded petitioner had been worked up for possible dystonia, but did not himself diagnose petitioner with dystonia. Dr. Lancaster also notes that dysautonomia does not logically explain dystonia.

Id. at 34-35.

Referring to Dr. Steinman's chart entitled "Symptoms Relevant to Opinion of Autoimmune Dysautonomia" (Ex. 94), Dr. Lancaster notes Dr. Steinman lists a series of symptoms as being relevant to his diagnosis of autoimmune dysautonomia. Dr. Lancaster states

he takes a different approach than Dr. Steinman by listing petitioner's most prominent symptoms and findings during the few months after petitioner's vaccination on August 23, 2009. Dr. Lancaster says petitioner's treating doctors listed these symptoms in their notes in the end of 2009 and the videos extensively document these symptoms. Dr. Lancaster classifies these symptoms as either plausibly related or not related to the autonomic nervous system. Id. Of the 16 symptoms[130] Dr. Lancaster lists (combining several of them to reach a total less than Dr. Steinman's total number of petitioner's symptoms), only two symptoms are potentially autonomic but could have many possible causes: (1) lightheadedness, dizziness; and (2) vomiting, poor GI motility. Id. at 36.

In conclusion, Dr. Lancaster states petitioner had diverse symptoms after flu vaccination including rhabdomyolysis, from which she quickly recovered, but her rhabdomyolysis was not related to her other subsequent symptoms. Id. He continues petitioner later reported symptoms for which doctors could not find a physiologic cause. These symptoms included numbness and weakness, fluctuating foreign accent syndrome, periods of speech arrest, seizure-like events, fluctuating memory difficulties, and a highly abnormal gait (e.g., ability to walk backward or sideways but not forward, making wildly jerking movements when walking). Later, petitioner developed periods of lightheadedness and difficulty eating. Id. Months later, petitioner reported symptoms suggestive of myasthenia gravis, but Dr. Lancaster notes objective evidence does not support this diagnosis. Id. at 36-37. Dr. Lancaster continues by stating the diagnosis of autonomic failure does not explain most of petitioner's symptoms and could not possibly account for her foreign accent syndrome, memory problems abnormal gait, numbness or weakness. Id. at 37.

Dr. Lancaster notes that the results of autonomic tests on petitioner conducted within a reasonable temporal interval after petitioner's flu vaccination do not support the diagnosis of autonomic failure. Her only abnormal gastric emptying study was conducted over two years after the vaccination. Moreover, a well-respected autonomic expert, Dr. Yan-Go, did a thorough evaluation of petitioner and concluded petitioner's autonomic function was intact. Id.

Dr. Lancaster reiterates that petitioner most likely has conversion disorder, which accounts much better for her most disabling symptoms, i.e., foreign accent syndrome, inability to walk forward (but ability to walk backward or sideways or to run), seizure-like events, bizarre gait consistent with astasia-abasia, confusion, and language disruption. He states multiple treating doctors suspected petitioner has conversion disorder. Dr. Lancaster notes that the medical records do not support a diagnosis of petitioner having an autoimmune disorder that would cause autonomic dysfunction, i.e., she did not have GBS or AAN. He continues that reliable scientific evidence is lacking to associate flu vaccine with a poorly specified autonomic

[130] The 14 symptoms Dr. Lancaster lists as not plausibly related to the autonomic nervous system are: unusual jerking gait; ability to run but not walk; ability to walk backward or sideways but not forward; tongue paralysis; limb paralysis; spitting food out of her mouth before swallowing; memory difficulties; confusion; inability to speak, speech arrest, quiet whispered speech; seizure-like events; unusual postures/dystonia; response to sensory tricks; prolonged dystonic posturing, speech arrest with tilt-table testing in the setting of normal blood pressure; and response to chelation therapy, hyperbaric chamber without oxygen, "drops" from Dr. Buttar; and improvement with exercise. Ex. H, at 35-36.

dysfunction. Moreover, reliable scientific evidence is lacking that any component of flu vaccine mimics an antigen specific to the autonomic nervous system. Therefore, Dr. Lancaster concludes that Dr. Steinman's proposed logical chain of causation between flu vaccine and petitioner's symptoms fails on multiple levels. Id.

On February 20, 2015, respondent filed the expert report of Dr. J. Lindsay Whitton, a virologist and immunologist whose practice is devoted to research. Ex. Z. Respondent filed Dr. Whitton's CV as Exhibit AA. Dr. Whitton received his M.B. Ch.B. (the United Kingdom equivalent of an M.D.) in 1979 and his Ph.D. in herpes virus transcription in 1984. Id. at 1. He is Professor in the Department of Immunology and Microbial Science at Scripps Research Institute. Id. He is a fellow in the American Academy of Microbiology, elected in 2011, and in the American Association for the Advancement of Science, elected in 2012. Id. He is a member of the following associations: American Association of Pathologists, American Association of Immunologists, American Society of Virology, and American Society of Microbiology. Id. He is on the editorial boards of the following journals: Journal of Virology, Virology (for which he is editor), BMC Microbiology (core reviewer), and Molecular Therapy. Id. at 1-2. He declined becoming editor of the Journal of Virology because he was editor for Virology. Id. at 2. He has authored or co-authored 185 articles (id. at 2-13); he has had 27 international lab trainees (id. at 19).

Dr. Whitton describes his role is to evaluate the biological plausibility of the mechanism(s) that Dr. Steinman uses to explain how flu vaccine could cause petitioner's signs and symptoms. Ex. Z, at 1. In the context of that purpose, Dr. Whitton states he has not read all the medical records but is familiar with the general facts of the case. In assessing Dr. Steinman's analysis, Dr. Whitton cautions that he focuses on the disease Dr. Steinman claims petitioner has but does not necessarily agree that Dr. Steinman's diagnoses are correct. Id.

Having read Dr. Steinman's three expert reports at that point (Exs. 65, 92, and 108), Dr. Whitton states they lack a consistent and clear explanation for how flu vaccine can cause the various diseases at issue in this case because Dr. Steinmann invokes essentially the entire immune system, i.e., innate immunity, T-cells, and other antibodies, yet appears to focus on a very specific immune response against "HFFK-like amino acid sequences." Ex. Z, at 2.

Dr. Whitton summarizes Dr. Steinman's analysis as follows:

1. Petitioner developed rhabdomyolysis (Ex. 65, at 1 and elsewhere);
2. Petitioner developed a form of GBS (Ex. 65, at 9);
3. Petitioner developed autoimmune autonomic ganglionopathy ("AAG"), resulting in autoimmune dysautonomia (Ex. 65, at 1 and elsewhere);
4. Flu vaccine administered August 23, 2009 caused all of the above (Ex, 65, at 1 and elsewhere); and
5. Molecular mimicry directed against "HFFK-like" amino acid sequences that flu vaccine and petitioner's myelin basic protein ("MBP") share, which is a component of most nerve cells, is the mechanism underlying the above diseases (Ex. 65, at 1, 10-15).

Id.

Dr. Whitton says he is intimately familiar with the theory of molecular mimicry, having been a laboratory colleague of Dr. Robert Fujinami in 1984 when he with their shared mentor Dr. Michael Oldstone popularized the concept of molecular mimicry in an article in the journal Science. In that article, Drs. Fujinami and Oldstone posited that hepatitis B virus could trigger an immune response that cross-reacted with a protein in the central nervous system ("CNS"), causing disease in the host. The central precept of molecular mimicry is that foreign material must be sufficiently different from the host to break immunologic tolerance, i.e., trigger an immune response in the person, but also sufficiently similar to allow those immune responses to cross-react against the person, causing autoimmune disease. Id.

Dr. Whitton agrees with Dr. Steinman who wrote that molecular mimicry is mainstream science, pointing to its being an explanation for how early versions of rabies vaccine, made in the brain tissue of rabbits or suckling mice, and administered for 21 days to humans, could induce cross-reactive responses, as anti-CNS antibodies, including anti-MBP antibodies, indicated in the patients' spinal fluid. Id. at 3. Dr. Whitton notes, however, that molecular mimicry does not cause many human diseases and, interestingly, the incidence of autoimmune disease has risen in locales in which infections have become far less frequent. Id.

Dr. Whitton states Dr. Steinman argues flu vaccine and the host protein MBP share a short homology, i.e., an HFFK-like motif (Ex. 65, at 10).[131] Id. Dr. Steinman contends the sequence FFKN in flu vaccine triggers an adaptive immune response, i.e., antibodies and T-cells, to enable the vaccinee, should she encounter flu virus, to fight if off by attacking the equivalent sequence in the viral protein. Id. at 3-4. But Dr. Steinman argues those antibodies and T-cells would also attack the HFFK-like sequence in MBP through molecular mimicry, causing neurological disease. Id. at 4.

Besides invoking the adaptive immune system, i.e., antibodies and T-cells, Dr. Steinman invokes the innate immune system constituting a less specific immune response that in general precedes the adaptive immune response and also contributed to petitioner's various diseases (Ex. 65, at 16-17). Dr. Whitton writes that, as far as he can tell, Dr. Steinman mentions the innate immune system in order to discuss molecules called toll-like receptors ("TLRs") which Dr. Steinman states are important in GBS. Id. Dr. Whitton notes that one of the articles Dr. Steinman cites in support of his thesis of molecular mimicry being vital in causing autoimmune diseases says infections may prevent autoimmune diseases. Thus, Dr. Whitton concludes that the significance of molecular mimicry in inducing the vast majority of autoimmune diseases remains highly speculative. Id.

Dr. Whitton goes through petitioner's symptoms in discussing her rhabdomyolysis. Ex. Z, at 5. On September 2, 2009, petitioner developed a sore throat, fevers, and a runny nose (Ex. 22, at 56) and later felt weak and dizzy, causing her to go to Loudon Hospital ED on September

[131] In Exhibit 65, at 10, Dr. Steinman writes, "Influenza viruses, like many other viruses, shares molecular similarities with one of the major myelin protein[s], myelin basic protein . . . specifically with an HFFK like motif." He continues, "The motif FFKN is shared between influenza and myelin basic protein." Ex. 65, at 11.

12, 2009 (Ex. 5, at 6). In the days before she was admitted to the hospital on September 12, 2009, petitioner had been doing daily training runs (Ex. 5, at 5). Blood work on September 12, 2009 showed elevated creatine phosphokinase, an enzyme in muscle cells. Its presence in petitioner's blood showed she had muscle cell damage over the prior few days. The hospital discharged petitioner on September 14, 2009 with a diagnosis of mild rhabdomyolysis, i.e., destruction of skeletal muscle cells. Dr. Whitton disagrees with Dr. Steinman's opinion that flu vaccine on August 23, 2009 caused petitioner's rhabdomyolysis (Ex. 65, at 1).

Dr. Whitton is puzzled as to how Dr. Steinman's theory of molecular mimicry between HFFK-like sequences applies to flu vaccine causing rhabdomyolysis. Dr. Whitton inquires what does Dr. Steinman think is the flu vaccine's proposed target protein, which presumably should be a protein in skeletal muscle cells. Id. Dr. Whitton asks where is the FFKN sequence that Dr. Steinman claims the flu vaccine-induced response attacked? Dr. Whitton notes that rather than explain this lapse, Dr. Steinman cites four articles, but the first involves flu virus and its relevance is unclear. The second is complicated because the patient was on statins when he received flu vaccine and rhabdomyolysis is a rare side effect of statins. The third and fourth papers discuss rhabdomyolysis in association with GBS, an association well-established but generally with the muscle disease occurring together with or after the onset of GBS's neurologic signs and symptoms. Dr. Whitton points out that when petitioner had rhabdomyolysis, she did not have any neurologic complaints. Her neurologic examination was normal (Ex. 5, at 7 and 10). When she did later complain of neurologic symptoms, Johns Hopkins neurologists attributed them to anxiety. Id.

Dr. Whitton gives two alternate explanations for petitioner's rhabdomyolysis which are far more likely than the flu vaccine. First, on September 2, 2009, petitioner developed sore throat, fever, and a runny nose. He states many viruses can cause myositis and rhabdomyolysis. Secondly, petitioner is an enthusiastic runner and, when she developed rhabdomyolysis, she was actively training for a 5K race. Dr. Whitton notes that exercise is a known cause of rhabdomyolysis. He says strenuous exercise most commonly triggers rhabdomyolysis in people who do not usually exercise, but it can occur unexpectedly even in habitual exercisers. Id.

Dr. Whitton, without opining whether petitioner had GBS since that is not the purpose of his expert report, notes that Dr. Steinman's provision of a Table and Figure on pages 12 and 13 of Exhibit 65 purportedly to show the importance of the FFKN sequence in GBS does not pertain to GBS, a disease of peripheral nerves, but to MS, a disease of central nerves. Id. at 7. Moreover, Dr. Whitton notes that Dr. Steinman relies on immune responses to myelin basic protein, which is not the target of the autoimmune response in GBS. Gangliosides are the target. Thus Dr. Whitton says Dr. Steinman does not provide a clear hypothesis to explain how flu vaccine causes GBS generally or in the instant case. Id. Dr. Whitton also notes that Dr. Steinman on page 11 of Exhibit 65 highlights two specific MBP sequences: HFFK and FFKN. Id. at 7-8. Dr. Whitton states the more loosely defined Dr. Steinman's target sequence is, the more likely it exists by chance in any given protein. Id. at 8.

Dr. Whitton states that humans have only about 20 different amino acids. The average protein contains about 500 amino acids, and there are about 30,000 proteins in the human

genome. Dr. Whitton says it is inevitable that a scientist will find identical sequences and multiple homologies. Id. Using a protein sequence search engine, Dr. Whitton found 50 human proteins (including MBP) containing the sequence FFKN. Id. at 9. Thus, the identification of FFKN or any other short sequence of amino acids in viral proteins and human proteins is predictable. Id.

As for Dr. Steinman's invoking the innate immune system in GBS in his analysis, and the increase of TLRs in the blood cells of people, Dr. Whitton comments that Dr. Steinman relies on what Dr. Whitton calls a "dreadful" study as well as a third-rate paper. Id. Dr. Whitton notes that a wide array of inflammatory stimuli activate TLRs. Id. Moreover, Dr. Whitton points out that antibodies, not T-cells, mediate GBS, which is why plasma exchange is the primary therapy for most GBS cases. Id. at 10.

Moving on to Dr. Steinman's thesis that vaccines can trigger AAG, Dr. Whitton takes issue with the papers upon which Dr. Steinman relies in making this statement. Id. Two of the four papers state vaccination may be associated with (but do not say "cause") autoimmune ganglionopathy, without providing support, while the other two papers do not mention vaccination. Id. Dr. Whitton then asks how Dr. Steinman's theory of HFFK-like molecular mimicry applies to AAG. Id. at 11. Dr. Steinman is unclear as to whether innate immunity or adaptive immunity causes AAG. Dr. Whitton would subscribe to an adaptive immunity analysis of AAG in which antibodies against acetylcholine receptors are present, but is puzzled why flu vaccine would trigger their production. If this were so, Dr. Whitton states acetylcholine receptors would have to contain the FFKN sequence, but there is no evidence that they do or even that petitioner has antibodies to acetylcholine receptors, for which Dr. Steinman noted petitioner had not yet been tested (Ex. 65, at 21). Id. Dr. Whitton notes that the ganglionic AChR antibodies found in many AAG patients recognize a specific subunit of ganglionic AChR which is a protein that does not contain HFFK. Dr. Whitton asks how Dr. Steinman's HFFK-like hypothesis explains how flu vaccine can cause AAG and says that it does not. Id.

In conclusion, Dr. Whitton says the evidence Dr. Steinman provides to support his opinion that flu vaccine administered on August 23, 2009 was responsible for petitioner's signs and symptoms is extremely weak. Dr. Whitton regards Dr. Steinman's invocation of molecular mimicry as implausible whether in the context of rhabdomyolysis, GBS, or AAG. He reiterates that petitioner's viral infection (sore throat, etc.) is much more likely than flu vaccine to have caused her rhabdomyolysis. Id. He notes that exercise could also have been a factor in causing her rhabdomyolysis. Id. at 12. Dr. Whitton says Dr. Steinman's molecular mimicry hypothesis fails to explain how HFFK-like sequences can cause rhabdomyolysis. Dr. Whitton notes that Dr. Steinman's focus on an FFKN-driven autoimmune response against MBP in causing GBS is invalid because gangliosides, not MBP, are GBS's target. Dr. Whitton criticizes Dr. Steinman's use of molecular mimicry between HFFK-like sequences between flu vaccine and human protein in explaining how flu vaccine caused AAG because the most common target protein in AAG does not contain HFFK or FFKN. Dr. Whitton concludes that Dr. Steinman's entire thesis requires flu vaccine to cause an adaptive, i.e., antibody or T-cell, immune response against

FFKN, but no one has ever made this finding even though Dr. Whitton says it would be relatively simple to determine. Id.

On June 1, 2015, petitioner filed Dr. Steinman's third supplemental expert report in which he comments on the videos that Dr. Lancaster reviewed in his expert report, and also responds to the expert reports of Dr. Lancaster (Ex. H) and Dr. Whitton (Ex. Z). Ex. 118. Dr. Steinman asserts that petitioner had many symptoms of autoimmune dysautonomia within 10 to 20 days after her August 23, 2009 flu vaccination. Dr. Steinman also asserts that petitioner's onset of myasthenia gravis "in hindsight" was about 20 days after that flu vaccination.[132] Id. at 1. Dr. Steinman summarizes Dr. Sheean's medical records and diagnoses, emphasizing that petitioner's having received IVIG treatments at the time she was tested for signs of myasthenia gravis impaired the test results. Id. at 2.

Dr. Steinman responds to Dr. Whitton's expert report (Ex. Z) first. Ex. 11, at 4. Dr. Steinman defends referring to myelin basic protein in patients with GBS. Id. at 6. He criticizes Dr. Whitton for not being licensed to practice medicine in the United States and for not having practiced medicine for decades. Id. Dr. Steinman states he will not react to Dr. Whitton's assertions about the medical records since Dr. Whitton chose not to read the complete set of records. Id.

Dr. Steinman then attacks Dr. Whitton's opinion that petitioner's viral illness of early September 2009 is the more likely cause of her rhabdomyolysis than the August 23, 2009 flu vaccination based on the ground that the type of viral infection petitioner had is unknown. Id. at 7-8. To support his thesis that an unknown virus cannot be the cause of rhabdomyolysis, Dr. Steinman quotes verbatim an excerpt from a decision[133] 10 years ago by another special master in another case in which that other special master says an unknown virus cannot be an alternate cause. Id. at 8.

Dr. Steinman seems unaware that another special master's decision does not bind the undersigned. Hanlon v. Sec'y of HHS, 40 Fed. Cl. 625, 630 (1998), aff'd, 191 F.3d 1344 (Fed. Cir. 1999) (special masters are not bound by their own or other special masters' decisions, or those of the Court of Federal Claims, except in the same case). In the almost 28 years of the undersigned's experience as a special master, the undersigned has never seen a medical expert attempt to support his position by citing a legal decision in another case instead of on medical literature and/or his own experience.

[132] Dr. Steinman's opinion that petitioner had myasthenia gravis changed over time. At the hearing, he distanced himself from the diagnosis. Fifteen months after the hearing, petitioner filed an amended petition on September 15, 2017, in which she alleges inter alia that flu vaccine caused her myasthenia gravis, but she follows that statement with a disclaimer that this is not Dr. Steinman's "favorite diagnosis" but Dr. Sheean's diagnosis and Dr. Steinman puts a "high value" on Dr. Sheean's opinion. Am. Pet. at ¶ 60.

[133] Torday v. Sec'y of HHS, No. 07-372V, 2009 WL 5196163 (Fed. Cl. Spec. Mstr. Dec. 10, 2009). Dr. Steinman was petitioner's neurologic expert in Torday. The issue in Torday was whether petitioner's flu vaccination or his preceding upper respiratory illness caused petitioner's GBS. Dr. Steinman testified that not all viruses cause GBS which persuaded the then-Chief Special Master to rule the vaccination caused petitioner's GBS.

Dr. Steinman's point is that a virus whose identity is unknown cannot be considered the cause of an illness that follows it. The undersigned rejects that point. To accept it would be to say since the upper respiratory infection petitioner in this case had in early September 2009 before she had rhabdomyolysis is unknown, respondent must prove all upper respiratory infections can cause rhabdomyolysis or the undersigned cannot find that petitioner's upper respiratory illness caused her rhabdomyolysis.

Dr. Steinman does not insist on such rigid proof for himself in his own expert reports that petitioner's flu vaccination caused her rhabdomyolysis. He relies solely on a couple of case reports, one of which involved someone who was taking a statin, which is itself a risk factor for rhabdomyolysis. Moreover, Dr. Steinman does not refrain from relying on articles about GBS, an illness petitioner did not have, as support for his opinion that petitioner had autonomic nervous system disease based on his "inference" she had GBS despite her normal neurological test results for GBS. Dr. Steinman is inconsistent in what he demands of respondent's expert yet allows for himself. Further in Dr. Steinman's response to Dr. Whitton's expert report, Dr. Steinman engages in a lengthy dispute about Dr. Whitton's understanding of GBS. But since petitioner never had GBS, this is not an issue in the case.

Dr. Steinman then goes on to say that a viral peptide of just five amino acids within a stretch of 12 amino acids can induce clinical signs of EAE with paralysis in mice. Ex. 118, at 14. EAE is experimental allergic encephalomyelitis.[134] Experimental allergic encephalomyelitis is "an animal model for acute disseminated encephalomyelitis in which the characteristic pathophysiology and clinical signs of this disease are produced by immunization of an animal with extracts of brain tissue or with myelin basic protein together with Freund adjuvant; it is transferable by adoptive transfer of lymphocytes but not by serum."[135] Encephalomyelitis is a disease involving inflammation of the brain and spinal cord.[136] But petitioner never had inflammation of her brain or spinal cord. Her brain MRIs were normal and her lumbar puncture showed her CSF (cerebrospinal fluid) was normal. Petitioner is not a mouse. The undersigned finds it irrelevant that Dr. Steinman can make mice ill with inflammation of their brains and spinal cords with a mere homology of five amino acids within 12 amino acids, plus Freund adjuvant, particularly since petitioner did not have GBS, which is a peripheral neuropathy which does not include inflammation of the brain or spinal cord. Multiple sclerosis, which is Dr. Steinman's specialty, does involve inflammation of the brain and spinal cord.

Dr. Steinman reiterates that there are six amino acids with close structural homology between flu A hemagglutinin in the 2009 Fluzone vaccine which cross-react with myelin basic protein. Id. at 15. In answer to Dr. Whitton's criticism that proof does not exist that flu vaccine induces a host response to the FFKN sequence, Dr. Steinman protests that absence of evidence does not mean evidence is absent. Id. at 16. But that is exactly what it means. A protestation

[134] Dorland's at 586.
[135] Dorland's at 614. Freund adjuvant is "a water-in-oil emulsion incorporating antigen, in the aqueous phase, into lightweight paraffin oil with the aid of an emulsifying agent. On injection, this mixture . . . induces strong persistent antibody formation." Id. at 32.
[136] Dorland's at 613.

that absent evidence does not mean that some time, some place, somewhere, evidence might be produced. This is speculation. Dr. Steinman is proficient at speculating, but short on proof.

Turning to Dr. Lancaster's expert report, Dr. Steinman disagrees that petitioner has conversion disorder. Id. at 17. Dr. Steinman protests that Dr. Lancaster has not met or treated petitioner. But then Dr. Lancaster has seen the contemporaneously recorded videos which means he saw what petitioner was manifesting which is the crux of Dr. Steinman's diagnosis of petitioner with autoimmune autonomic neuropathy and autonomic ganglionopathy. It is true that neither Dr. Lancaster nor Dr. Steinman is petitioner's treating physician. Dr. Steinman states there was no triggering event[137] for petitioner's having conversion disorder. He thinks doctors mistook petitioner's POTS and myasthenia gravis[138] for anxiety.

Although Dr. Lancaster considers petitioner's symptoms of profound language disturbance, including foreign accent syndrome and periods of being unable to speak, inability to walk forward but ability to walk backward and respond to sensory tricks, reported ataxia, tremors, and cognitive complaints are localized to the central nervous system, and not to the autonomic nervous system, Dr. Steinman disagrees. He thinks the inability to walk forward at times but the ability to walk backward are classic manifestations of certain movement disorders connected to the autonomic nervous system. Dr. Steinman refers to the "Inside Edition" interview of petitioner and writes, "It is hard to imagine that someone would concoct these symptoms, or that it is a conversion disorder." Id. at 18. Dr. Steinman attributes petitioner's foreign accent syndrome to an impairment to the muscles involved with speech that myasthenia gravis causes. Id. at 19. He thinks her foreign accent is merely the perception of her listeners. Id.

Dr. Steinman agrees petitioner did not have classic GBS, but he thinks she had an inflammatory neuropathy in which dysautonomia is the dominant feature. Id. at 21. Dr. Steinman cites the Suarez article[139] in support of petitioner's having an inflammatory neuropathy in which dysautonomia is the dominant feature. He thinks idiopathic autonomic neuropathy is at one end of the spectrum and GBS is at the other. He agrees with Dr. Lancaster that a specific autoantibody test for AAG should have been done, but he disagrees that the failure to do this test means petitioner did not have AAG. Id. Nowhere, except for a brief mention of the "Inside Edition" video, does Dr. Steinman describe the numerous videos and comments Dr. Lancaster made concerning them. Only on January 13, 2016 did petitioner file Dr. Steinman's fourth supplemental report in which he discusses the videos. Ex. 114.

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[137] Dr. Steinman has forgotten that stress can trigger conversion disorder. Dorland's at 549. See also, Functional Neurologic Disorders/Conversion Disorder, MAYO CLINIC, https://www.mayoclinic.org/diseases-conditions/conversion-disorder/symptoms-causes/syc-20355197 (last visited Mar. 1, 2019) ("The cause of functional neurologic disorders is unknown. The condition may be triggered by a … reaction to stress….").

[138] Dr. Steinman wrote this report before he decided petitioner's having myasthenia gravis was not his "favorite" diagnosis. See Am. Pet. at ¶ 60. At the hearing, he abandoned the diagnosis of myasthenia gravis and just stated petitioner had autoimmune autonomic neuropathy and vagus nerve injury.

[139] Guillermo A. Suarez et al., Idiopathic Autonomic Neuropathy: Clinical, Neurophysiologic, and Follow-up Studies on 27 Patients, 1994 NEUROLOGY 1675 (1994); Ex. 132.

On September 4, 2015, respondent filed the first supplemental expert reports of Dr. Lancaster and Dr. Whitton. Exs. RR and SS.

Dr. Lancaster notes that he reviewed additional exhibits 11-38 including the records of Dr. Elmer Chang, dated November 2, 2011, who reviewed petitioner's negative autonomic studies at UCLA (upper endoscopy, CT of the abdomen and pelvis, gastric emptying study, and esophageal manometry (Ex. 114, at 1-5)). Ex. RR, at 1. Dr. Lancaster also notes that, on January 29, 2015, Dr. Sheean saw petitioner and she told him she was taking one Mestinon a day, which Dr. Lancaster thought unusual because Mestinon lasts only four hours. Id. at 2. Dr. Lancaster also notes, on February 10, 2015, petitioner had a negative result of a repetitive nerve stimulation study, involving the ulnar, accessory, radial, and median nerves, at rest and after exercise to see if she had evidence of a neuromuscular junction disorder (Ex. 116, at 13-18). Id. at 3.

Dr. Lancaster emphasizes that myasthenia gravis does not affect sensation, cognition, or autonomic function. Id. at 4. Patients with myasthenia gravis may have weakness and decreased muscle tone, but they do not become spastic or have abnormal movements. Dr. Lancaster states that the likelihood that a patient with generalized myasthenia gravis, as petitioner alleges, would be negative for MuSK antibodies, negative for AChR antibodies, and have negative repetitive stimulation studies is about 2 percent. Id. Dr. Lancaster's opinion is that petitioner's diagnosis of myasthenia gravis is very unlikely to be correct, particularly in light of petitioner's other symptoms that are very unlikely to have a physiological basis. Id.

Dr. Lancaster criticizes the chart Dr. Steinman created as a timeline of petitioner's symptoms (Ex. 117) purporting to show symptoms of myasthenia gravis or AAN because petitioner did not have either disease. Id. at 4-5. Dr. Lancaster also criticizes the symptoms Dr. Steinman attributes to these disorders because they could never reasonably be attributed to them. Id. at 5. Dr. Lancaster states spastic jerking limb movements are not symptoms of myasthenia gravis. Dr. Lancaster says spasticity and jerking movements occur due to a central nervous system disorder, and both involve excessive movements and increased muscle tone. But myasthenia gravis affects the nerve-muscle junction in the peripheral nervous stem, causing fatigue, weakness, and in extreme myasthenia gravis, decreased muscle tone. Id. Dr. Lancaster notes that Dr. Steinman attributes petitioner's talking in one-word answers to myasthenia gravis, leading to respiratory failure. But Dr. Lancaster heard petitioner speaking in the videos and petitioner was not having respiratory failure in them. Her inability to speak occurred while she was breathing normally. Moreover, Dr. Lancaster found particularly noticeable petitioner's dramatic improvement in speech when she started to run a 5K race, which is exactly the opposite of what someone with myasthenia gravis would experience in which sudden exertion would worsen shortness of breath and make speaking even harder. Dr. Lancaster notes petitioner's apparent foreign accent that could not be due to AAN or myasthenia gravis. Id.

Dr. Lancaster states that tingling in hands and feet would involve somatic nerves, not autonomic nerves, and Dr. Steinman should not have attributed those symptoms to AAN. Moreover, myasthenia gravis does not cause sensory symptoms or affect sensory nerves. Id. Dr. Lancaster describes petitioner's difficulty walking during the months after her August 23, 2009

flu vaccination as consisting of wild lurching movements that actually require excellent balance to perform. He saw extensive video evidence of petitioner's gait problem. He labels this phenomenon astasia-abasia, which is a psychogenic movement disorder and would not occur with either myasthenia gravis or autoimmune autonomic neuropathy. Id. Dr. Lancaster states that someone with autoimmune autonomic neuropathy might become lightheaded with exertion but would not engage in wild, lurching movements, and if the patient did, he or she would find them more difficult to perform than normal walking. Dr. Lancaster says myasthenia gravis might cause fatigue, which would result in a patient with that disease walking slowly or needing to rest frequently, but not engaging in wild lurching movements, which require a lot of energy to perform. Id. He notes that at the time petitioner performed her most prominent gait symptoms, she also ran a 5K race, which resulted in sudden resolution of her symptoms with running. Dr. Lancaster says this would be even more inconsistent with petitioner having AAN or myasthenia gravis. He says a patient with weakness from myasthenia gravis would not suddenly feel better with running but would have even worse shortness of breath and need to stop. Id. A patient with autoimmune autonomic neuropathy causing autonomic dysfunction who became lightheaded with walking would probably collapse if he or she attempted to run. Id. at 5-6. In autonomic nervous system disease, a patient can barely maintain adequate blood pressure to perfuse the brain while walking. Id. at 6. The additional blood flow demands of running would cause collapse.

Dr. Lancaster notes Dr. Steinman attributes petitioner's complaints of shooting, tingling pains in her lower extremities to both AAN and myasthenia gravis. Dr. Lancaster says myasthenia gravis causes painless weakness and he would not attribute pain of any kind to myasthenia gravis. He says AAN affects autonomic ganglia, not pain fibers, and would not cause pain either. Id.

Having watched petitioner's videos, Dr. Lancaster comments that petitioner's being unable to move or speak was not attributable to myasthenia gravis. He notes petitioner did not have any symptoms of myasthenia gravis, e.g., ptosis, weakness, ocular movements, or respiratory weakness. Petitioner also appeared to have fluctuating consciousness during these events, also not attributable to myasthenia gravis. A patient in myasthenia gravis crisis to the extent of not being able to move his or her limbs would typically need to be placed on a ventilator immediately or he or she would die. Id.

Dr. Lancaster says that petitioner's report at Johns Hopkins of lower extremity weakness and numbness was not due to myasthenia gravis. He notes that myasthenia gravis does not cause numbness. The weakness in myasthenia gravis is typically bulbar (involving muscles of the face and neck) more than the limbs and does not ascend. He notes that skilled neurologists at Johns Hopkins evaluated petitioner and did not think her complaints had a physiological cause and they did not diagnose her with myasthenia gravis. Id.

Dr. Lancaster states that Dr. Steinman attributed petitioner's inappropriate laughter to autoimmune autonomic neuropathy. Dr. Lancaster questioned how a disorder of the autonomic nervous system could cause inappropriate laughter. Dr. Lancaster thinks Dr. Steinman's statement is implausible. Dr. Lancaster notes that Dr. Steinman incorrectly attributes numerous

other symptoms to myasthenia gravis: relief of speech symptoms by placing a hand on her chin, dystonia, foot tapping, head bobbing, improvement of speech with singing, walking better backward than forward, speaking with a foreign accent, contracting her arm and face when speaking, etc. Id.

Dr. Lancaster notes that Dr. Steinman attributes dystonic posturing and apparent dystonic symptoms to autoimmune autonomic neuropathy. However, dystonia is not a symptom of autoimmune autonomic neuropathy because dystonia comes from the brain and not from the autonomic nervous system. In addition, Dr. Lancaster does not think petitioner actually had dystonia. In summary, Dr. Lancaster states Dr. Steinman's attribution of petitioner's numerous symptoms to autoimmune autonomic neuropathy or myasthenia gravis is illogical and incorrect. Id.

Dr. Lancaster states it is unclear to him whether Dr. Steinman is asserting flu vaccine caused petitioner autoimmune autonomic neuropathy or some other less clearly defined disorder when Dr. Steinman asserts petitioner has autoimmune dysautonomia due to an autoimmune inflammatory neuropathy as a result of flu vaccination. Id. at 7.

Dr. Lancaster takes issue with Dr. Steinman's assertion that petitioner's symptoms of myasthenia gravis began within 20 days of vaccination. Dr. Lancaster says petitioner had rhabdomyolysis possibly due to an infection or running about 20 days after vaccination and nine days after a flu-like illness. Then petitioner reported numerous neurologic symptoms, e.g., numbness, weakness, shaking, cognitive complaints, tongue paralysis, foreign accent syndrome, about 25 days post-vaccination. Dr. Lancaster says these symptoms were generally not symptoms of myasthenia gravis, especially foreign accent syndrome, shaking, and cognitive complaints. Expert neurologists at Johns Hopkins did not think petitioner had a physiologic basis for her complaints. From September to December 2009, petitioner's primary problems were seizure-like events, speech disruption (generally foreign accent syndrome), and a bizarre lurching gait that improved with running. Dr. Lancaster notes that petitioner's videos "provide abundant evidence of these symptoms," and he detected nothing in these videos to make myasthenia gravis a plausible diagnosis. Id.

Dr. Lancaster notes that from January 2010, petitioner's symptoms shifted to those resembling autonomic failure, but that diagnosis has scant objective evidence. Id. He states only in about 2012 did petitioner have symptoms resembling myasthenia gravis, years after the vaccination at issue. Even in 2012, objective testing of petitioner for myasthenia gravis was repeatedly negative and she most likely does not have it. Id.

Dr. Lancaster says he agrees that Dr. Sheean's testing and Dr. Gee's earlier testing in 2012-2013 do not show any objective evidence of myasthenia gravis. Dr. Lancaster does not agree with Dr. Steinman's opinion that IVIG treatments might account for petitioner's negative test results. He notes petitioner's testing on two occasions for AChR and MuSK antibodies were negative. Her repetitive stimulation studies were negative twice as well. He states IVIG might dilute her immunoglobulins slightly, but there is no evidence and basis to think that IVIG would result in negative test results. Dr. Lancaster states patients with real autoantibodies have levels at

an order of magnitude higher than control patients who do not have myasthenia gravis, which IVIG cannot normalize. Moreover, Dr. Lancaster states IVIG would not normalize petitioner's electrophysiology tests. He emphasizes that Dr. Gee tested petitioner when she was not on IVIG treatments. Dr. Lancaster concludes the most logical reason petitioner tested negative for myasthenia gravis both before and after she started receiving IVIG is that she did not have myasthenia gravis. Id.

Dr. Lancaster disagrees with Dr. Steinman's theory that a similarity between flu protein and myelin basic protein underlies petitioner's symptoms which Dr. Steinman diagnoses as myasthenia gravis and autoimmune autonomic neuropathy. Ex. RR, at 8. Dr. Lancaster states he agrees with Dr. Steinman that myelin basic protein is a major component of insulating cells of both the central and peripheral nervous systems, but autoimmunity to myelin basic protein has never been demonstrated to be important for the pathophysiology of myasthenia gravis, which involves autoantigens that are clearly neuromuscular junction proteins on skeletal muscles cells and do not have significant amounts of myelin basic protein. Id. Therefore, Dr. Lancaster says, it is implausible that autoimmunity to myelin basic protein would cause myasthenia gravis. Moreover, Dr. Lancaster states that autoimmunity to myelin basic protein has never been shown to cause autonomic failure and, therefore, Dr. Steinman's reliance on an explanation for autonomic failure based on autoimmunity to myelin basic protein is scientifically unreliable. The theory of myelin basic protein autoimmunity is most accepted for the model of MS, which is a completely different disease which fails to explain petitioner's wide-ranging constellation of symptoms. Id.

Dr. Lancaster states petitioner's preceding viral infection or her running caused her rhabdomyolysis. He finds the lack of a specific cause for the virus unsurprising due to the diversity of viral infections. He thinks the flu vaccine is a less likely cause than the viral infection. He notes petitioner's rhabdomyolysis was self-limited and cured with appropriate treatment. It does not explain petitioner's subsequent diverse symptoms for which no physiologic cause was established. Id.

Dr. Lancaster finds interesting Dr. Steinman's discussion of the animal model of encephalomyelitis in discussing how molecular mimicry between proteins in flu vaccine and myelin basic protein may be related to petitioner's illness. Id. However, if flu vaccine affected petitioner in this way, she should have had acute disseminated encephalomyelitis ("ADEM") or MS, not myasthenia gravis or autonomic autoimmune neuropathy. Id. Regarding Dr. Steinman's statement that some autonomic fibers are myelinated, Dr. Lancaster asks why autoimmunity to myelin basic protein selects autonomic fibers. In Dr. Steinman's animal model, this autoimmunity targeted the brain, which has abundant myelin. Autoimmune autonomic neuropathy is selective for autonomic ganglia because ganglionic AChR (the target antigen) is selectively expressed in autonomic ganglia. However, Dr. Lancaster says there is no evidence that autoimmunity to myelin basic protein causes any autonomic or neuromuscular junction disorder in people. Id.

Dr. Lancaster notes that Dr. Steinman's diagnosis of petitioner's problem has changed from his initial expert report to include myasthenia gravis and autonomic failure, but they do not

account for most of petitioner's symptoms (numbness, foreign accent syndrome, lurching gait, altered consciousness, paralysis) during the first three months after her flu vaccination. Id. at 8-9. Now, Dr. Steinman seems to be proposing in Exhibit 118, at 18, that petitioner's gait is a form of dystonia, adding a third diagnosis, which is wrong. Id. at 9. Dr. Lancaster says that dystonia is a central nervous system disorder, directly contradicting Dr. Steinman's theory that petitioner's symptoms are due to myasthenia gravis and autoimmune autonomic neuropathy. Dr. Lancaster states the autonomic nervous system has nothing to do with dystonia. The autonomic nervous system controls involuntary processes, e.g., sweating, pupillary constriction, heart rate, and blood pressure. It does not control complex limb movements or gait. Id.

Dr. Lancaster asks how does Dr. Steinman prove flu vaccine causes dystonia, now that Dr. Steinman added dystonia to myasthenia gravis and autoimmune autonomic neuropathy as diagnostic of petitioner's disorders? Dr. Lancaster says none of these three diseases is explainable by hypothetical autoimmunity to myelin basic protein and all three involve distinct regions of the nervous system. Id. If petitioner had brain lesions, why would she not have MS and ADEM as well as dystonia?

Dr. Lancaster says petitioner's foreign accent syndrome cannot be related to myasthenia gravis. The video shows petitioner shifting in and out of a British accent with a strong clear voice. He says myasthenia does not do this. He adds that petitioner's reported focal paralysis of her tongue is also not related to myasthenia. Dr. Lancaster says that if petitioner had autoimmunity to myelin basic protein, she would have peripheral and central nervous system effects because myelin basic protein is so widespread. She would not have focal autonomic dysfunction. Id.

Dr. Lancaster states that the reason no one tested petitioner for antibodies to myelin basic protein is that they are not useful for diagnosing any neurologic disease. Id. at 10. Dr. Lancaster criticizes Dr. Steinman's remark that the proof that petitioner does not have conversion disorder is her response to treatment (Ex. 118, at 18). Dr. Lancaster says that he personally witnessed petitioner having a miraculous response from Dr. Buttar's hyperbaric chamber without oxygen, chelation, and TD-DMPS drops that Dr. Buttar placed on petitioner's forearms. He says, "It is completely implausible that these treatments would affect dystonia, AAN, or MG—let alone cure all three diseases in a matter of seconds." Id. His explanation for petitioner's "cure" is conversion disorder plus the repeated suggestions that she would respond to these treatments. Id.

As for Dr. Steinman's criticism of Dr. Lancaster's saying petitioner has conversion disorder because Dr. Lancaster is not a psychiatrist, Dr. Lancaster responds that a neurologist's role is to exclude organic etiologies. Dr. Lancaster says he spent many hours reviewing petitioner's entire medical record and had the benefit of directly observing petitioner's symptoms for many hours on video. The videos were probably the equivalent of 20 typical office visits. He says there is no plausible physiological basis for petitioner's most striking symptoms and she has conversion disorder. Id.

Dr. Lancaster says petitioner does not have dystonia. He defines dystonia as a movement disorder characterized by sustained or intermittent muscle contractions causing abnormal tone or

movements. Id. Initially, petitioner's symptoms resembled GBS, not dystonia. Id. at 11. But the Johns Hopkins neurologist concluded that petitioner's symptoms did not have a physiological basis. Afterwards, petitioner developed an abnormal gait and unusual speech patterns. He states a careful review of the videos shows these symptoms were not of dystonia. Dr. Lancaster says vocal dystonia causes a strained or hoarse voice, not speaking with a British accent. Her lurching gait is characteristic of a psychogenic movement disorder, not dystonia. Her wild lurching without falling is characteristic of psychogenic movement disorder. Sudden onset at maximal severity is also characteristic. Dr. Lancaster notes that petitioner's movement problem came about after doctors told her she did not have GBS. These movements faded as her symptoms focused more on autonomic and apparently myasthenic symptoms. Dr. Lancaster notes that when confronted by "Inside Edition," her symptoms suddenly recurred, which is also characteristic of psychogenic movement disorders. Dr. Lancaster states that petitioner complained of many symptoms that dystonia does not cause or with which dystonia is not associated. Id. These symptoms include alteration in consciousness, sensory symptoms (numbness), profound weakness or paralysis, and speaking with a foreign accent. Id. Dr. Lancaster says that if petitioner had dystonia, vaccination did not cause it. Most cases of dystonia have no known cause. Id.

Dr. Lancaster concludes that the long period of time between vaccination and symptoms suggestive of myasthenia gravis make vaccination as the cause very unlikely. Id. at 11-12. Vaccinations do not cause autoimmunity to myelin basic protein, and autoimmunity to myelin basic protein would not explain focal autonomic failure, myasthenia gravis, or dystonia. Id. at 12. He states vaccination did not cause petitioner's symptoms. Id.

Dr. Whitton notes in his supplemental expert report (Ex. SS) that he obtained a Ph.D. after his medical degree, and he pursued a career specializing in virology and immunology. Ex. SS, at 1. He has received five National Institute of Health grants to focus on viruses, immunology, and disease. Id. at 2. Dr. Whitton states that Dr. Steinman failed to address many of the questions Dr. Whitton raised in his first expert report (Ex. Z). Ex. SS, at 2. Dr. Whitton says that Dr. Steinman identifies the proposed cellular target for rhabdomyolysis as nerve cells (Ex. 118, at 5). Ex. SS, at 3. Dr. Whitton notes if nerve damage caused the death of muscle cells in rhabdomyolysis, petitioner should have had preceding or concurrent neurologic signs and symptoms, but petitioner's neurological examination was normal (Ex. 5, at 7 and 10).

As for Dr. Steinman's position that petitioner's preceding viral infection does not count as a cause of her rhabdomyolysis because no one knows the identity of the viral infection, Dr. Whitton responds that doctors do not identify the great majority of viral infections. Id. Dr. Whitton notes that the risk of GBS increases after undiagnosed infections. Id. As for Dr. Steinman's position that exercise could not explain petitioner's rhabdomyolysis because she is an experienced runner, Dr. Whitton reiterates there is a case report in which an experienced runner, after a minor change in her training routine, came down with rhabdomyolysis. Id. at 4. Dr. Whitton states that notably absent from Dr. Steinman's response is evidence that flu vaccine can cause rhabdomyolysis. Id.

Dr. Whitton takes issue with Dr. Steinman's contention that myelin basic protein is an important target in GBS because Dr. Steinman relies on an article that is over three decades old in support of that proposition. Id. To disprove Dr. Steinman's new claim (Ex. 118, at 14) that the peptide sequence FYKNLI (in Fluzone vaccine) is homologous to the sequence FFKNIV in myelin basic protein and, thus, the immune system sees the F and Y amino acids as mimics, Dr. Whitton looked at the data from Dr. Steinman's laboratory and found that if someone takes a peptide with the FFKN motif and replaces either of the Fs with a Y, antibodies can no longer see the peptide efficiently. Id. at 4-5. Thus, because the FFK core amino acids are missing from the flu peptide sequence of FYKNLI in Fluzone vaccine, the antibodies would not efficiently recognize them and would not be a compelling molecular mimic of the sequence FFKNIV in myelin basic protein. Id. at 6-7.

Dr. Whitton notes that Dr. Steinman has still not explained how he thinks flu vaccine causes AAG. Id. Dr. Steinman admits that the HFFK-like motif has nothing to do with producing antibodies against acetylcholine receptors. Id. at 9. Dr. Whitton characterizes Dr. Steinman's theories as "jumbled." Id. at 10. Dr. Whitton asks if Dr. Steinman's "current" hypothesis is that flu vaccine triggered two completely distinct sets of molecular mimicking responses. The FYKNLI in flu vaccine induced one response which hypothetically cross-reacted with the FFKNIV sequence in myelin basic protein, even though, as Dr. Steinman published, the F to Y difference dramatically altered any potential cross-reactivity of the two sequences. Supposedly, this imagined cross-reactive response would have caused both rhabdomyolysis and GBS even though myelin basic protein is not an important target antigen in GBS. Moreover, Dr. Whitton asks if Dr. Steinman is proposing that flu vaccine induced a second, completely different immune response, which an unidentified sequence in flu vaccine induced and which then cross-reacted with an unidentified protein on nerve cells (either AChR or something in myelin) to cause AAG? Dr. Whitton says he does not understand what hypotheses Dr. Steinman is proposing. Id.

On January 13, 2016, petitioner filed Dr. Steinman's fourth supplemental expert report. Ex. 141. In the first 20 pages of Exhibit 41, he responds to Dr. Whitton's supplemental expert report (Ex. SS). In the last six pages, he responds to Dr. Lancaster's comments on videos dated October 17-22, 2009 (Ex. H). Dr. Steinman lists the cases in which he and Dr. Whitton have debated at hearing the merits of Dr. Steinman's thesis that HFFKNIV is a molecular mimic. Dr. Steinman mentions that a number of cases have settled and states, "I have no idea of the impact of my expanded analysis in the strategic decision to settle, but it certainly appears that Respondent is aware of much that I shall present here." Ex. 141, at 1. Dr. Steinman devotes the next two pages to his many disagreements with Dr. Whitton, indicative of a long history of professional feuding.

Dr. Steinman then reveals a "remarkable" feature of the Fluzone vaccine petitioner received in 2009, i.e., it contains H_1N_1 viruses capable of eliciting anti-ganglioside antibodies, one of the known antibodies with which some forms of GBS are associated. Id. at 6. Dr. Steinman concludes that petitioner's 2009 flu vaccination not only had a component capable of inducing anti-myelin basic protein antibodies, but also anti-ganglioside antibodies. All of Dr.

Steinman's discussion in this section of his report assumes petitioner had GBS. But the fact that flu vaccine can cause GBS does not mean it did cause GBS in this case if petitioner did not have GBS. In response to a question in one of the undersigned's orders whether Dr. Steinman thinks petitioner has idiopathic torsion dystonia (dystonia musculorum deformans), Dr. Steinman replies petitioner has both autoimmune dysautonomia due to autoimmune autonomic neuropathy and myasthenia gravis. Id. at 20-21. He notes dysautonomia also appears in myasthenia gravis. Id. at 21.

Dr. Steinman then proceeds to comment on videos dated October 17-22, 2009 upon which Dr. Lancaster commented in Exhibit H, which respondent filed one year earlier on January 13, 2015.

For the October 17, 2009 video (#409_0239_01), petitioner was preparing to run a race and reported she could not drink water without triggering convulsions. Dr. Lancaster states in his expert report that this does not correspond to a neurological disorder in his experience. Dr. Steinman comments he has not heard of this in his experience either. He interprets petitioner's statement as manifesting clear dysarthria, i.e., drinking involves muscles needed for speech and swallowing and, therefore, drinking water worsened petitioner's muscle weakness in her neck, causing her head to shake. Id.

In another video (#409_252_01), petitioner is lying on a couch being fed during an attack and reporting difficulty moving her tongue. Her then-husband discusses how moving food around with her tongue causes her to seize. Id. Dr. Lancaster states this is inconsistent with petitioner's claim that her tongue is paralyzed and appears most consistent with a psychogenic, non-epileptic seizure-like movement because she was jerking bilaterally while remaining conscious. Id. at 21-22. Dr. Steinman says he cannot comment because he would need a concomitant EEG and the best would be a video EEG. Id. at 22.

In another video (#409_254_01), petitioner is lying on her back and speaking with a slurred speech pattern while shaking. Dr. Lancaster considers this most consistent with a psychogenic disorder. Dr. Steinman says he cannot comment because he would need a concomitant EEG and video monitoring. Id.

In another video (#409_0257_02), petitioner continues to relate the story of her initial illness, describing seizure-like episodes, being able to walk backward but not forward, being able to walk but not talk at times or talk but not walk at other times. Id. She also describes relieving vocal symptoms by putting her hand on her chin. Dr. Lancaster comments this is inconsistent with Dr. Steinman's theory that an autoimmune condition caused petitioner's symptoms because a real autoimmune brain disease causing neurologic deficits would not resolve and recur within a period of seconds and would not respond to sensory tricks. Dr. Steinman comments that cues can modulate successfully neurologic deficits whether autoimmunity triggered them or not, and that a patient can learn from experience. Id.

In another video (#409_0257_03), petitioner explains how all her symptoms resolve when she runs, but all her symptoms immediately recur when she stops running. Dr. Lancaster says this is inconsistent with an autoimmune brain disease. Petitioner also reports convulsions

and blacking out when she stops running. Dr. Steinman comments, "It is good to listen to the patient, but I find it difficult to integrate these comments with symptoms." He also reiterates he would like to see this when petitioner has concomitant video and EEG monitoring. Id.

For the October 18, 2009 video (#409_0278_01), petitioner is taking some kind of liquid medicine from a spoon, then swallowing pills, and then drinking liquid several times, all without difficulty. Id. At 4:22 p.m., she has a non-epileptic seizure-like event, which Dr. Lancaster depicts as a psychogenic seizure. During the event, her body shakes but Dr. Lancaster notes she is able to put her drink down properly. Then she closes her eyes, which Dr. Lancaster says is a predictive factor for a non-epileptic event. Id. Dr. Lancaster also notes that petitioner carefully positions herself to avoid injury during the event. Id. at 22-23. Dr. Steinman's only comment is, "I would like to see all these activities during concomitant EEG monitoring." Id. at 23.

In another video (#409_0298_01), petitioner's then-husband describes petitioner as having a seizure-like event and describes her episode of rhabdomyolysis, then losing the ability to move or speak, and her suspected GBS. He says petitioner's team of doctors were stumped and later she could whisper, but not talk, which the neurologists could not explain. He says two psychologists saw petitioner for one hour and she was discharged from Fairfax Hospital and went to Johns Hopkins and told the doctors there that her diagnosis was GBS. Johns Hopkins gave petitioner Ativan and when she woke, she spoke perfectly normally. She later had a miraculous recovery of her strength, doing 20 laps and a little dance in her room, but then went into what her then-husband described as bigtime seizures. A physical therapist examined petitioner and thought she had dystonia, telling petitioner of medication and sensory tricks. Petitioner was discharged with instructions to follow up with a therapist and seemed to get her speech and walking ability back with benzodiazepine. He says she would have seizures if she had any stimuli such as a dog barking or a plane flying overhead, or if more than one thing was going on at a time. Her seizures were attributed to the benzodiazepines. Petitioner and her then-husband spoke on the phone with someone who thought her problem was autoimmune and that benzodiazepines were treating her autoimmune disease. Dr. Lancaster says the idea that benzodiazepine would treat an autoimmune disease has no medical basis. He also says that these so-called triggering events for epileptic seizures are extremely unlikely. Dr. Lancaster views these events as psychogenic and non-epileptic, and not as epileptic seizures. Dr. Steinman's comment is his familiar, "I would need to see an objective situation with EEG monitoring and video in order to analyze the events." Id.

Dr. Steinman then states his view on all these videos: "In general for all the discussions and video sessions, my inclination is to watch and listen thoughtfully, but really I would need more objective evidence with EEG and video in order to make any pronouncements." Id.

Dr. Steinman then moves onto the videos taken October 19, 2009. Video #409_0313_01 depicts petitioner arriving at Dr. Buttar's clinic, lying down in the clinic and communicating with gestures, but not talking. Id. Stan Kurtz thinks petitioner should not speak and they should carry petitioner. Id. at 23-24. Petitioner has rhythmic head shaking when a nurse asks her a question. Id. at 24. She covers her face with her hands. Dr. Lancaster comments this did not look like epilepsy but as another non-epileptic psychogenic event. Dr. Steinman continues his refrain:

"My inclination is to watch and listen thoughtfully, but really I would need more objective evidence with EEG and video in order to make any pronouncements."

In another video (#409_0318_01), petitioner's then-husband and Stan Kurtz discuss how anything, e.g., eating or seeing a floor pattern, can trigger a seizure in petitioner. Petitioner feels the blood move from her head down to her stomach when she eats. They think she is too ill to answer questions. Dr. Lancaster comments that the fact that eating or a floor pattern can trigger these events is another factor supporting his diagnosis of psychogenic events rather than epileptic seizures. Id. Dr. Steinman repeats his mantra: "My inclination is to watch and listen thoughtfully, but really I would need more objective evidence with EEG and video in order to make any pronouncements."

In another video (#409_0325_01), petitioner receives food by spoon, then stares and Stan Kurtz says, "She's seizing." Petitioner responds somewhat to stimuli (such as a hand near her eye) and then hyperventilates a bit when the event stops. Dr. Lancaster says this event is most consistent with another psychogenic, non-epileptic event. Petitioner retches but does not appear to be actually vomiting. Dr. Steinman repeats his mantra: "My inclination is to watch and listen thoughtfully, but really I would need more objective evidence with EEG and video in order to make any pronouncements." Id.

In another video (#409_0326_01), petitioner makes more retching noises but does not apparently vomit. She clenches her arms and has another shaking event. Dr. Buttar arrives and petitioner lies on the bed with her eyes closed as Dr. Buttar speaks. Dr. Buttar plans to insert an IV, but Stan Kurtz says that when petitioner "is in this state any activity can set her off." He gives as examples a rug pattern, making eye contact, or hearing two voices at the same time. Petitioner has head shaking after she squeezes Dr. Buttar's hand on request. Dr. Lancaster says this is all much more consistent with psychogenic events than with epilepsy. Dr. Steinman repeats his mantra: "My inclination is to watch and listen thoughtfully, but really I would need more objective evidence with EEG and video in order to make any pronouncements." Id.

In another video (#409_0346_01), petitioner is being transferred to a wheelchair and retching. She also had rhythmic head shaking during this while her eyes are closed. Id. at 25. Nevertheless, Dr. Steinman repeats his mantra: "My inclination is to watch and listen thoughtfully, but really I would need more objective evidence with EEG and video in order to make any pronouncements." Id.

In another video (#409_0382_01), Stan Kurtz reports that petitioner spoke very well for three hours after earlier hyperbaric chamber therapy. Stan Kurtz says the hyperbaric therapy makes petitioner speak normally. The video shows petitioner out from the chamber communicating with gestures. She whispers in a very normal voice that she can wiggle her toes and states in a whisper that she had a seizure just in her toes. Dr. Buttar plans more therapy with mist and chelators. Id. Dr. Steinman comments, "I myself am quite skeptical about hyperbaric therapy and chelators." Id. He repeats his mantra by saying "my inclination is to watch and listen thoughtfully, but really I would need more objective evidence with EEG and video in order to make any pronouncements." Id.

In another video (#409_0401_01), petitioner speaks normally while in the hyperbaric chamber. She reports she is speaking normally and she drinks water without difficulty. Dr. Steinman comments with the exact comment he made before about his skepticism about hyperbaric therapy and chelators, but his inclination is to "watch and listen thoughtfully" and he needs to have "more objective evidence with EEG and video."

Dr. Steinman moves on to videos dated October 22, 2009. In videos #409_0541_01, #409_0542_01, and #409_0544_01, petitioner has electrodes placed on her head for a qEEG. Id. He agreed with Dr. Lancaster that an epilepsy specialist should review the entire data of the study. Id. at 26. He admits he is not a board certified EEG specialist.

In all, whereas Dr. Lancaster in Exhibit H, at 11-21, describes and comments upon 178 videos, Dr. Steinman in Exhibit 141, at 21-25, describes and comments upon only 17 videos. Most of his comments on these videos is that he would watch and listen thoughtfully, and he could not make any other comment until he had more objective evidence with EEG and video monitoring. He ignores the fact that petitioner has had numerous EEGs, all of which were normal.

Dr. Steinman concludes that none of petitioner's doctors excluded the diagnosis of autoimmune dysautonomia due to autoimmune autonomic neuropathy or of myasthenia gravis and, therefore, she cannot have a conversion disorder. Id.

On June 20, 2016 (which was after the hearing held on June 14-17, 2016), the undersigned issued an Order that Dr. Steinman and Dr. Whitton give their opinion on whether the negative test for flu A and B virus antigen on September 12, 2009 means the flu vaccination petitioner received on August 23, 2009 was ineffective and whether that affected their expert opinions.

On July 14, 2016, respondent filed Dr. Whitton's second supplemental expert report. Ex. YYY. Dr. Whitton states that the goal of testing for viral antigens is to determine if a patient had a current viral infection. Id. at 1. The medical records show that on September 2, 2009, petitioner had a sore throat, fever, and a runny nose. On September 12, 2009, she went to Loudon Hospital ER where she was diagnosed with rhabdomyolysis. Since viral infections quite commonly cause rhabdomyolysis, the doctor did a throat swab to look for streptococcal infection, and a nasopharyngeal wash to look for the presence of flu virus (Ex. 51, at 138). Both tests were negative. Id. The way to test for the effectiveness of a vaccine is to look for pathogen-specific antibodies with a blood draw. Also, a doctor would test for pathogen-specific T-cells with a blood draw. Id. Thus, to test for a vaccine's effectiveness, a doctor would not look for antigens; instead the doctor would look for antibody and T-cell responses to antigen. Id. at 2. Therefore, petitioner's negative testing for flu A and B virus antigen on September 12, 2009 did not mean the flu vaccination petitioner received was ineffective. Id. She was never tested for its effectiveness. Id. Dr. Whitton stated his opinion on the case has not changed. Id.

On July 14, 2016, petitioner filed Dr. Steinman's fifth supplemental expert report. Ex. 181. His opinion is similar to Dr. Whitton's and his opinion remains unchanged. Id. at 1.

On October 14, 2016, after petitioner filed the medical records of MedStar Georgetown University Hospital (Ex. 180) where the EMTs took petitioner on June 15, 2016 when she sank to the floor while growling during the hearing, petitioner filed Dr. Steinman's sixth supplemental expert report. Ex. 191. Dr. Steinman states petitioner was making loud inspiratory noises from the seating area while he was in court. Id. at 1. He heard and observed her inspiratory stridor. Petitioner wrote on a pad her medication for myasthenia gravis, i.e., Mestinon, after Dr. Steinman asked if she took the medicine. Dr. Steinman states he was at petitioner's side during the 16 minutes it took for the paramedics to arrive, but Dr. Lancaster "did not offer any assistance." Id. This is a cheap shot. None of the doctors in the room moved to assist petitioner when she sank to the floor while growling. The undersigned instructed Dr. Steinman to attend to petitioner since he was her expert. Only then did Dr. Steinman get up from his chair to assist her. There was no need for Dr. Lancaster to offer any further assistance.

Dr. Steinman then pastes into his sixth supplemental expert report the neurological record from MedStar Georgetown and stresses the importance of the mention at Ex. 183,[140] at 5, of petitioner having ptosis. Ex. 191, at 2. He writes ptosis is a manifestation of myasthenia gravis and states it cannot be faked. However, Dr. Steinman failed to paste into his sixth supplemental expert report the further comment of Dr. Brian Barry, the neurologist, on the same page (Ex. 183, at 5), stating that petitioner had bilateral mild to moderate ptosis which could be overcome spontaneously with upgaze, and which was not worse with fatigue.

Dr. Steinman comments on the finding of an elevated anti-GAD antibody. Ex. 191, at 8. Dr. Steinman states he has studied and published on GAD itself and anti-GAD antibodies in type 1 diabetes and multiple sclerosis. He says anti-GAD antibodies have been reported in conditions that include dystonia and neuromyotonia.[141] He cites three references[142] and says petitioner has elements of what these references describe, referring to petitioner's "quite extraordinary neurologic functions." Id. Dr. Steinman says petitioner's anti-GAD antibodies are "profoundly important in unraveling her case." Id. He ran a BLAST search for homologies between GAD and elements of the 2009 Fluzone vaccine whose components are: A/Brisbane/59/2007 (H_1N_1)-like virus, A/Brisbane/10/2007 (H_3N_2)-like virus, and B/Brisbane/60/2008-like virus. Id. He says he found a "remarkable" molecular mimic between the A/Brisbane/10/2007 (H_3N_2)-like virus and GAD.[143] Id. Although he admits that petitioner was not tested for anti-GAD antibodies recently, he says the results of the test provide objective evidence for her early

[140] Petitioner filed the complete record of June 15, 2016 at MedStar Georgetown, including the ED record, as Ex. 180. Dr. Steinman, in his sixth supplemental expert report, is referring only to the neurological portion of that record which is Ex. 183.

[141] Neuromyotonia is "myotonia caused by electrical activity of a peripheral nerve, characterized by stiffness, delayed relaxation, fasciculations, and myokymia." Dorland's at 1267. Myotonia is "dystonia involving increased muscular irritability and contractility with decreased power of relaxation." Id. at 1226. Myokymia is "a benign condition marked by brief spontaneous tetanic contractions of motor units or groups of muscle fibers, usually adjacent groups of fibers contracting alternately." Id. at 1223.

[142] The undersigned identifies and discusses these three references in the section entitled "Medical Literature and Other Filings."

[143] Dr. Steinman discovered at the hearing that he had mistakenly analyzed the components of the trivalent flu vaccine of the 2010-2011 flu season, and not the components of the trivalent flu vaccine petitioner received in 2009. Tr. at 530-35.

symptom onset. Id. at 10. He thinks the results of the test prove petitioner does not have conversion disorder. Id. Dr. Steinman concludes that based on this recent information, he adds a further assertion, i.e., that petitioner's flu vaccination likely triggered autoimmunity to GAD (glutamic acid decarboxylase). Id. at 12.

Dr. Steinman engages in a paean to Dr. Sheean, reciting his credentials: In 2005, 2006, Dr. Sheean won the Senior Faculty Teaching Award at the University of California, San Diego.[144] Id. Dr. Sheean joined the faculty of the University of California San Diego in February 1998 and serves as Director of the Neuromuscular Division. The website continues and states Dr. Sheean does both teaching and clinical research. He has used botox to treat spasticity and movement disorders and maintains a demanding schedule of clinical activities, including several weekly clinics, consisting of five EMG clinic sessions, one neuromuscular clinic, one ALS clinic, one botulinum toxin injection clinic, and one botulinum toxin review and assessment clinic. Id. at 5-6. Dr. Sheean treats patients with a variety of neuromuscular conditions, including myasthenia gravis and peripheral neuropathy. Id. at 6. In July 2003, Dr. Sheean was named Director of the Neurology Outpatient Clinic at Perlman Ambulatory Care Center in La Jolla. In July 2004, Dr. Sheean helped establish the ALS Center at the UCSD Medical Center in Hillcrest and served as co-Director for the first two years of operations. As Director of the Neuromuscular Fellowship Program, Dr. Sheean is responsible for the selection and training of fellows. All his clinics incorporate teaching for EMG Fellows and neurology residents. He trains neurology residents in EMG and nerve conduction during their four-month rotation through the service in their first year. One of the busiest clinics is devoted to using botulinum toxin injections to treat focal dystonia, minifacial spasm, spasticity, and migraine. Dr. Sheean provides tutorials and case presentations in the Clinical Core Clerkship course. He gives an annual lecture and demonstration on nerve conduction and EMG in the basic neurology course. He received the Clinical Teaching Award for 1999-2000. Id. Neither Dr. Steinman nor the UCSD website mentions that Dr. Sheean is not board certified in neurology. See supra, n.101.

On December 16, 2016, respondent filed Dr. Whitton's third supplemental expert report (Ex. ZZZ) and Dr. Lancaster's second supplemental expert report (Ex. FFFF). Dr. Whitton states that Dr. Steinman's initial reports suggest that petitioner suffered from rhabdomyolysis, GBS, and autonomic ganglionopathy due to responses to a short amino acid sequence of myelin basic protein. Ex. ZZZ, at 1. Dr. Steinman proposed this link to myelin basic protein by relying

[144] A UCSD website depicting Dr. Sheean's name and his publications, and describing his title as "Associate Physician Diplomate, Medicine" at Health Sciences. UCSD Profiles: Geoffrey Sheean, UC SAN DIEGO, https://profiles.ucsd.edu/geoffrey.sheean (last visited Mar. 19, 2019). This website lists Dr. Sheean's "concepts" derived automatically from his publications. These research areas include: muscle spasticity; botulinum toxins, type A; neuromuscular agents; electromyography; torticollis; etc. for a total number of 119 concepts. Among these research areas, myasthenia gravis is not listed. https://profiles.ucsd.edu/display/180912/Network/ResearchAreas (last visited Mar. 19, 2019). Dr. Sheean is no longer listed on the UCSD faculty website that Dr. Steinman used for his description of Dr. Sheean's past accomplishments: https://neurosciences.ucsd.edu/faculty/Pages/geoffrey-sheean.aspx (last visited Mar. 20, 2019). Clicking on that cited page brings up this statement instead: "The page you're looking for doesn't exist." School of Medicine, UC SAN DIEGO SCHOOL OF MEDICINE, https://medschool.ucsd.edu/Pages/PageNotFoundError.aspx?requestUrl=https://medschool.ucsd.edu/faculty/Pages/geoffrey-sheean.aspx (last visited Mar. 20, 2019).

on a homology between that protein and flu vaccine. However, at the hearing, Dr. Steinman realized he had erred because petitioner received a different vaccine than the one Dr. Steinman had analyzed. As a result, Dr. Steinman openly rejected the myelin basic link and relied instead on other vaccine sequences that he said were more important than the myelin basic protein sequences and which he claimed could cause disease by cross-reacting with two other central nervous system proteins (myelin oligodendroglial glycoprotein and 2,3CNPase). In Dr. Steinman's latest expert report (Ex. 191), he invokes another protein as a target for immune cross-reactivity, i.e., glutamic acid decarboxylase (GAD). Id.

Dr. Whitton notes petitioner displayed signs of distress at hearing, necessitating the phone call to paramedics. The medical records from MedStar Georgetown University Hospital note petitioner's inspiratory stridor resolved after a single dose of Mestinon. Dr. Whitton discusses a possible placebo effect of Mestinon because hospital personnel noted that petitioner's inspiratory stridor was inconsistent with myasthenia gravis. Id.

Dr. Whitton discusses Dr. Steinman's invoking a protein as a target for immune cross-reactivity called GAD and says Dr. Steinman seems to be proposing in Ex. 191, at 11, that petitioner suffers from stiff person syndrome. Ex. ZZZ, at 2. Dr. Whitton comments that Dr. Steinman put a lot of weight on Dr. Sheean's opinion, stressing repeatedly that he did so in part because Dr. Sheean was on the staff at Scripps, the same outstanding institution where Dr. Whitton works. As Dr. Whitton said at the hearing, to the best of his knowledge, Dr. Sheean does not have an appointment at or association with the Scripps Research Institute where Dr. Whitton works. (Apparently, Dr. Sheean has moved on professionally. He apparently is no longer on the faculty of UCSD. See supra, n.144.).

Dr. Whitton explains that GAD has two isoforms in humans: GAD65 and GAD67. Ex. ZZZ, at 2. Antibodies specific for both these isoforms are associated with type 1 diabetes and stiff person syndrome. Dr. Whitton says that Dr. Steinman in Ex. 191, at 11, appears to believe that petitioner has stiff person syndrome. Dr. Whitton notes that petitioner's detectable level of GAD antibodies in her blood may not be significant because her blood level was very low. He states this low level would not support the diagnosis of stiff person syndrome because, in stiff person syndrome, the anti-GAD antibody levels are usually high. Secondly, he notes that although anti-GAD antibodies are associated with stiff person syndrome does not mean they are always causal. Instead, anti-GAD antibodies may be the consequence of stiff person syndrome. Additionally, Dr. Whitton points out that petitioner received multiple IVIG infusions, which can lead to false blood test results. He further notes that 1-2 percent of the general population has anti-GAD antibodies. IVIG is prepared from a pool of about 1,000 blood donations and the constitution of the IVIG reflects the serological status of the donor population. Dr. Whitton expects that any preparation of IVIG would contain immunoglobulins from 10-20 donors who are positive for anti-GAD antibodies. Id.

Dr. Whitton moves on to Dr. Steinman's identification of two homologies between a vaccine protein, A/Brisbane/10/2007 (H_3N_2)-like virus, and human GAD. Id. at 3. Dr. Steinman states there are four identical residues out of one in one homology and five out of seven identical residues out of a second homology. But, Dr. Whitton says this applies only to mice who receive

pertussis toxin and a strong adjuvant, which differs dramatically from flu vaccination administered to humans. Moreover, Dr. Whitton notes that flu vaccine has never been shown to cause EAE in mice. Id. Dr. Whitton says that Dr. Steinman does not provide any evidence that an immune response to either of the GAD67 "homologies" actually occurs or is in any way harmful. Id. at 3-4.

Dr. Whitton states that short homologies are predictable and are not "remarkable" (as Dr. Steinman characterized them). Id. at 4. Dr. Whitton continues that flu virus HA protein is 329 amino acids in length, whereas human GAD67 protein is 594 amino acids in length. Dr. Whitton says it is not remarkable that there would be four out of five homologies or five out of seven homologies between them. Id. Dr. Whitton says that short homologies are not unusual. Id. at 5. He notes that viruses contain multiple proteins while the human proteome is thought to contain about 30,000 proteins. A scientist would not be surprised to find a very large number of short matches between the proteins of viruses and people merely by chance. Id. at 5-6.

To illustrate the fallacy of Dr. Steinman's thinking, Dr. Whitton did an experiment using the same vaccine sequence Dr. Steinman used and comparing it by a BLAST search against human albumin, a protein playing a key function in the bloodstream. Id. at 6. Human albumin at 609 amino acids is very similar in length to GAD67. He then shows that the vaccine protein had several homologies with albumin, but the data is limited and does not prove disease causation. Id. at 6-7. Dr. Whitton concludes that the mere presence of such homologies does not mean they have biological significance. Id. at 7.

In Dr. Lancaster's second supplemental expert report (Ex. FFFF), he states that stress may have triggered petitioner's stridor on June 16, 2016 during the hearing in this case. Ex. FFFF, at 1. He notes petitioner did not show any signs of myasthenia gravis during this episode. She was able to move air strongly, as Dr. Steinman later noted. Her ability to move air strongly was apparent from the loud sounds she generated while breathing, which required considerable respiratory muscle strength. Dr. Lancaster also notes that the MedStar Georgetown University Hospital records state petitioner had mild to moderate bilateral ptosis which looking up (i.e., up-gaze) could overcome and which did not fatigue. Dr. Lancaster states this signifies that petitioner's ptosis may not be due to myasthenia gravis. Id. She might instead have had eye closure weakness. The Georgetown neurologist Dr. Brian Barry who performed a physical examination on petitioner expressed doubts about whether petitioner's symptoms were physiological because, although she measured 4/5 strength, she dropped her arms one to two seconds after holding them up. Id. Dr. Barry noted petitioner had possible evidence of effort dependence, meaning it was questionable how hard petitioner was trying to generate full strength and questionable whether she had actual weakness. Id. at 1-2.

Dr. Lancaster also notes petitioner had negative inspiratory force, which means she was not weak in breathing. Id. at 2. Dr. Barry noted this negative inspiratory force was inconsistent with myasthenia gravis weakness. Id. Dr. Lancaster quotes the Georgetown Hospital notes on Dr. Gee's phone call with Dr. Barry, to the effect that petitioner had frequently been admitted to the hospital in California with low negative inspiratory force, which is effort dependent, but no neuromuscular cause was believed associated with these spells and her myasthenia was stable.

Dr. Gee also told Dr. Barry petitioner had been previously assessed for non-organic dystonia attributed to a flu vaccination. Dr. Lancaster stresses this is a very important observation, suggesting Dr. Gee believed petitioner's abnormal gait after flu vaccination was non-organic, i.e., did not have a physiological basis. This is another way of saying it is psychogenic. Id.

Dr. Lancaster notes that petitioner's GAD65 antibodies on August 4, 2016 were mildly elevated at 22.2 units. Id. at 3. The normal range is 0.0 to 5.0 units. However, in Dr. Lancaster's experience, a strong GAD65 response is generally measured as >250 units. Petitioner had normal test results for CK, copper, anti-thyroglobulin, thyroglobulin, TTG antibody, SSA antibody, SSB antibody, ceruloplasm, and MuSK antibody. All of petitioner's objective tests for myasthenia gravis were negative. Id. On physical examination, petitioner did not have stiff person syndrome. Another GAD65 antibody test was weakly positive at 11 units. Dr. Lancaster notes that the diagnosis of myasthenia gravis was not raised until well over a year after petitioner's flu vaccination. Myasthenia gravis would not explain her lurching gait (astasia-abasia), foreign-accent syndrome, ability to run but not walk, ability to walk backward, but not forward, trouble concentrating, seizure-like spells, etc. Dr. Lancaster says he agrees with Dr. Whitton's analysis of GAD65 antibodies. They are found in one to two percent of the general population. Petitioner's low-titer GAD65 antibodies could have come from her IVIG infusions. Id. Dr. Lancaster notes that Dr. Steinman's proposed homology between flu vaccine components and GAD65 resembles chance events. Id. at 4. Dr. Lancaster writes that people with type 1 diabetes frequently have GAD65 antibodies.

Dr. Lancaster writes stiff person syndrome is an acquired disease causing increased muscle tone. Stiff person syndrome patients have very strong GAD65 antibody responses. They do not have sudden attacks of abnormal movements that rapidly resolve as petitioner's abnormal movements rapidly resolved. Stiff person patients do not have foreign-accent syndrome, seizure-like events, and the ability to walk backward, but not forward. Dr. Lancaster writes petitioner did not have stiff person syndrome in the three months after her flu vaccination or at any time thereafter. Id. He says petitioner's rapid response to chelation therapy drops and other unconventional treatments Dr. Buttar gave her is entirely inconsistent with stiff person syndrome. Id. at 5. Dr. Lancaster writes that stiff person syndrome patients would not respond to Mestinon.

Dr. Lancaster says that a subset of patients with epilepsy have GAD65 antibodies, but it is unknown how the antibodies relate to the seizures. Id. Dr. Lancaster notes petitioner did not have cerebellar degeneration. Patients with cerebellar degeneration may have GAD65 antibodies. Patients with cerebellar degeneration have progressive nystagmus and ataxia of their limbs and gait. They have severe difficulty moving. These symptoms are static or slowly progressive. They do not suddenly come and go over minutes. A patient with cerebellar degeneration would not have a wildly lurching gait one minute and then run a race normally seconds later. If patients with cerebellar gait disorders attempted petitioner's wildly lurching gait shown in her videos, they would fall. He states the many hours of videos are the key to diagnosing petitioner with conversion disorder. Id.

Patients with encephalitis and opsoclonus-myoclonus may have GAD65 antibodies, but it is unknown if the antibodies cause any of these syndromes. In any event, the GAD65 antibodies are high titer. Petitioner did not have any of the syndromes associated with GAD65. Id.

Dr. Lancaster comments that ptosis can be faked (psychogenic pseudoptosis) just by lowering one's eyelids. Id. at 6. The Georgetown neurologist overcame petitioner's ptosis by having her look up. Dr. Lancaster states physiological ptosis would not be overcome with up-gaze. He also attributed petitioner's recovery when she took Mestinon to the placebo effect. He said there is no logical link between flu vaccination seven years previously and petitioner's recent measurement of GAD65 antibodies. Dr. Lancaster regards the IVIG infusions as the cause of these GAD65 antibodies. Id.

Dr. Lancaster questions the metamorphosis of Dr. Steinman's constantly shifting theories and diagnoses. Id. at 6. Dr. Lancaster states he is unsure of what Dr. Steinman's current theory of the case is on either an immunologic or clinical level. Dr. Steinman now seems to believe flu vaccine caused GAD65 antibodies and therefore her symptoms. Dr. Lancaster states there is no logical link between a vaccination administered seven years earlier and her recent measurement of GAD65 antibodies. Also, this is a completely different theory than Dr. Steinman's original theories on autoimmunity to myelin basic protein. He asks if Dr. Steinman believes both theories are true. Dr. Lancaster asks which specific type of autoantibody is supposed to have caused petitioner's foreign-accent syndrome, her ability to walk backward, but not forward, and her ability to run but not walk. Dr. Lancaster says there is no reliable medical evidence that flu vaccine can cause autoimmunity to either myelin basic protein or GAD65. Furthermore, there is no evidence that petitioner had autoimmunity to myelin proteins at all, let alone that this would have clinical significance. Now years after vaccination, she has a very low-titer GAD65 response, best explained by the IVIG infusions and unlikely to have any diagnostic significance. Id.

Dr. Lancaster notes that Dr. Steinman states he stands by his original opinions in this case, but his initial diagnosis was that petitioner had a form of autonomic neuropathy. Id. at 6-7. Dr. Lancaster states the autonomic nervous system controls unconscious or subconscious neurological functions, including the regulation of heart rate, the regulation of blood pressure, the constriction of pupils in response to light, sweating, etc. Id. at 7. The autonomic nervous system does not regulate gait or accent; neither does it cause seizure-like events. Moreover, autoimmune autonomic disorders such as autoimmune autonomic neuropathy have well-defined clinical features: orthostasis, fixed or unresponsive pupils, anhidrosis, gastrointestinal immobility, etc. Id. Dr. Lancaster recalls Dr. Steinman invoking the diagnosis of dystonia. Dystonia involves the pathways for controlling voluntary motor functions in the brain. Patients with dystonia have abnormal fixed postures or abnormal muscle contraction with movements. Dystonia does not cause foreign-accent syndrome or petitioner's wildly lurching gait (astasia-abasia). Dystonia comes from the brain and not the autonomic nervous system. Many forms of dystonia are considered of genetic origin and only a few cases are thought to be autoimmune, such as dystonia of anti-NMDAR encephalitis. Thus, Dr. Lancaster identifies that Dr. Steinman

introduced a second diagnosis with a completely different localization and pathophysiology with the diagnosis of dystonia. Id.

Dr. Lancaster notes that Dr. Steinman adds a third diagnosis to autonomic neuropathy and dystonia: myasthenia gravis which was only suggested one year after vaccination. Id. Myasthenia involves autoantibodies to the neuromuscular junction on the post-synaptic side, i.e., on the muscle side of the nerve-muscle communication. Dr. Lancaster stresses there is still no objective evidence that petitioner has myasthenia gravis. She was tested for acetylcholine receptor antibodies, MuSK antibodies, and with neurophysiology. The negative test results strongly suggest the diagnosis of myasthenia gravis is incorrect. Moreover, petitioner's symptoms after vaccination are inconsistent with a diagnosis of myasthenia gravis. He states a patient with active myasthenic weakness cannot run a 5K race, as petitioner did. Id.

Dr. Lancaster notes that Dr. Steinman adds a fourth diagnosis, i.e., a GAD65-associated syndrome. Dr. Lancaster wants to know which syndrome Dr. Steinman proposes: stiff person syndrome, cerebellar ataxia. Dr. Lancaster states petitioner does not have either. Even if she had those syndromes now, they would not explain her symptoms after vaccination since neither of these conditions causes someone to run but not walk, to walk backward but not forward, to speak with a foreign accent, or to have seizure-like events. Id. Neither syndrome would have the dramatic responses to placebo seen in the videos when Dr. Buttar rubbed chelation drops on petitioner's forearms. Id.

Dr. Lancaster states it would be entirely unprecedented for one patient to have so many presumably autoimmune disorders affecting so many distinct areas of her nervous system. Id. at 8. He says it would be even more remarkable that her condition is able to remit completely, sometimes in seconds, and leave no convincing objective evidence of its existence. By contrast, conversion disorder is quite common and easily accounts for petitioner's prominent symptoms after vaccination and those evolving over time. Id.

On May 16, 2017, petitioner filed Dr. Steinman's seventh supplemental expert report. Ex. 198. He thinks petitioner's collapse and stridor in the courtroom on the second day of the hearing was proof of her myasthenia gravis, a consequence of petitioner's being overdue for IVIG, and the stress of travel and of testifying in the case. Id. at 1-2. Dr. Steinman believes that petitioner's most recent May 2017 anti-GAD antibody test result of 37 proves she is undergoing an immunologic process rather than a passive transfer of anti-GAD antibodies through IVIG transfusions. Id. at 3. Dr. Steinman thinks that petitioner's anti-GAD antibodies explain the diagnoses of her myasthenia gravis, stiff person syndrome, intestinal pseudo-obstruction, and autonomic neuropathy. Id. Dr. Steinman says that even if petitioner's anti-GAD antibodies were a result not of her illnesses but of passive transfer from her IVIG treatment, she would not be receiving IVIG treatment but for her vaccine reaction, and therefore the anti-GAD antibodies are a sequela of her vaccine injuries. Id. at 4.

Dr. Steinman disagrees with Dr. Whitton's dismissal of the homology thesis between flu vaccine and human GAD that Dr. Steinman previously put forth. Id. He disagrees with Dr. Whitton's statement that short homologies are predictable. Id. at 7. Dr. Steinman notes that

petitioner "is a complex individual, as we all are." Id. at 18. He says that there are aspects of petitioner's responses in the videos that perplex him. He says, "Not everything in medicine is clear cut, easy to diagnose, black and white." Id. He relies on petitioner's neurologists "who made concrete diagnoses and prescribed powerful medicines." Id. He thinks petitioner's insurance company would not have paid for petitioner's IVIG treatments if IVIG were just a placebo. He thinks, "A placebo effect under such circumstances is too speculative" and undermines petitioner's neurologists while elevating the "speculative opinion" of respondent's experts. Id.

Dr. Steinman doubts that petitioner was feigning ptosis. Id. He recognizes that Dr. Lancaster relied on the videos in supporting his opinion that petitioner has conversion disorder, but Dr. Steinman says this is illogical because her treating physicians Dr. Cintron and Dr. Atiga did not opine that. Id. Dr. Steinman said Dr. Cintron knew about the bizarre aspects of the case and discussed sensory tricks but still did not think petitioner's illness was functional, i.e., non-organic. Id. at 19.

On May 17, 2017, petitioner filed her supplemental declaration. Ex. 205. She states that Dr. Gee told her that some of the spasms and tremors she manifested in her videos can be explained by a high GAD antibody. On May 17, 2017, the undersigned issued an Order for petitioner to file a statement from Dr. Gee indicating whether he believes petitioner had a high GAD in 2009 and, if she did, whether the high GAD antibody would cause some of the spasms and tremors she had in 2009 as shown in her videos. Moreover, the undersigned wanted to know if Dr. Gee thought petitioner had myasthenia gravis in 2009. Petitioner filed a status report stating Dr. Gee did not give her a statement to file.

On June 19, 2017, petitioner filed Dr. Steinman's eighth supplemental expert report. Ex. 206. Dr. Steinman writes that petitioner was not tested for high GAD antibody in 2009, but he thinks that her early-onset symptoms indicate that she very likely did have a high GAD antibody in 2009. Ex. 206, at 1. He notes that he previously attributed petitioner's early symptoms of tremors and spasms to myasthenia gravis. He attributes her other symptoms to high GAD as well. He writes that petitioner has been diagnosed (or a doctor considered whether to diagnose her) with many illnesses connected to anti-GAD antibodies: (1) stiff person syndrome; (2) intestinal pseudo-obstruction;[145] and (3) autonomic neuropathy. Id. He continues that petitioner's early onset of symptoms indicates: (1) myasthenia gravis; (2) intestinal pseudo-obstruction; and (3) autonomic neuropathy. Id. He opines that petitioner did have myasthenia gravis in 2009. Id. at 2. He believes that petitioner's 2009 flu vaccination likely triggered autoimmunity to GAD in petitioner. Id.

On July 18, 2017, the undersigned issued an Order asking Dr. Steinman to clarify his opinion since it shifted from an initial diagnosis of autoimmune autonomic neuropathy to explain petitioner's earliest symptoms to her having myasthenia gravis as the explanation for all her

[145] Intestinal pseudo-obstruction is "a condition characterized by constipation, colicky pain, and vomiting, but without evidence of organic obstruction; it is frequently a motor disorder." Dorland's at 1545.

symptoms. The undersigned gave petitioner the option to reopen testimony, amend her petition, or any other action. Petitioner chose to file an amended petition and post-hearing briefs.

On October 30, 2017, petitioner filed Dr. Steinman's ninth supplemental expert report. Ex. 208. He states it is not his opinion that all of petitioner's initial symptoms were related to myasthenia gravis. Id. at 3. He states GAD antibody helps explain all of petitioner's symptoms, including those relating to dystonia, gastroparesis, myasthenia gravis, and intestinal pseudo-obstruction. Id. He notes petitioner's GAD antibody tests ranged from 22.2 (August 4, 2016, Ex. 185, at 3), to 11 (August 14, 2016, Ex. 189, at 1), to 6 (January 13, 2017, Ex. 207, at 4), to 37 (May 2, 2017, Ex. 204, at 1). Id. He attaches a medical article in which respondent's expert Dr. Lancaster is a co-author (Ex. 209) as proof that therapeutic plasma exchange and immunosuppressive therapy lower levels of anti-GAD antibodies and improve clinical symptoms (without mentioning that the patient on whom the article focuses had a level of GAD autoantibody of 115,900IU/ml and, even after five sessions of therapeutic plasma exchange, had an anti-GAD antibody level of 3,970IU/ml). See Ex. 209, at 4. This level of anti-GAD antibody is profoundly higher than petitioner's range of 6 to 37. Dr. Steinman concludes that flu vaccine likely triggered petitioner's autoimmunity to GAD. Ex. 208, at 5.

On October 30, 2017, respondent filed the fourth supplemental expert report of Dr. Whitton (Ex. MMMM) and the third supplemental expert report of Dr. Lancaster (Ex. QQQQ). Dr. Whitton notes that Dr. Steinman agrees that IVIG infusions can passively transmit anti-GAD antibodies. Ex. MMMM, at 1. Both Dr. Whitton and Dr. Lancaster point out that petitioner's serum reflected anti-GAD antibodies at a low titer. Dr. Whitton finds Dr. Steinman's disagreeing because the lab test results indicate a "high" reference value to be disingenuous because anything above normal is reported as "high." In practice, Dr. Whitton points out, the extent of the elevation above normal is often expressed as "low" or "high" titer. Thus, slightly above normal, as were petitioner's lab results, would be referred to as "low titer," while a huge increase would be referred to as "high titer." Id. Petitioner's anti-GAD antibody titers between 6 and 37 were in the relatively low range, i.e., above normal, but not extremely high. Id. Dr. Whitton states the upper limit of normal is 5IU/ml for anti-GAD antibodies. Id. at 2. Thus, the lowest above normal reading petitioner could have would be 6, which she had on January 13, 2017. Stiff person syndrome patients have anti-GAD antibody titer levels that are very high, from 600IU/ml to 3,000IU/ml. Id.

Dr. Whitton emphasizes the changing diagnoses Dr. Steinman provides in his expert reports over time. Dr. Steinman's initial reports reflect his belief that petitioner had three illnesses: rhabdomyolysis, GBS, and autonomic ganglionopathy. Id. Dr. Steinman claims all three were responses to a homology between a short amino acid sequence in flu vaccine and myelin basic protein. At the hearing, Dr. Steinman discovered he erred and explicitly abandoned the homology of myelin basic protein and flu vaccine because petitioner received a different vaccine than the one he analyzed. Dr. Steinman then discarded the homology to myelin basic protein and relied on other vaccine sequences that he said were more important than the myelin basic protein homology and which Dr. Steinman asserted cross-reacted with myelin oligodendroglial glycoprotein and 2,3CNPase. In Ex. 191, Dr. Steinman added stiff person

syndrome to petitioner's conditions and asserted this resulted from immune cross-reactivity between a vaccine protein and GAD. In Ex. 198, Dr. Steinman asserts petitioner has myasthenia gravis, which his first report never mentions, but does not explain how flu vaccine triggers myasthenia gravis. Id.

Dr. Whitton points out that Dr. Steinman, in Ex. 198, did two identical BLAST searches to look for homologies and reported different outcomes. Id. at 4. Dr. Whitton says that comparing any two proteins of a reasonable length, e.g., 400-500 amino acids, would produce homology of 5/12. Id. at 6. Dr. Whitton disagrees with Dr. Steinman that homology proves molecular mimicry has occurred. Dr. Whitton notes that when a scientist injects a protein in a lab animal, the animal makes immune responses only to certain parts of the protein, not to each and every run of amino acids in the protein. Id. When a scientist finds a homology, he cannot assume that a foreign part of the homology triggers any immune response in the animal. Dr. Whitton states that proteins fold into complex three-dimensional structures and the relevant part of the host protein may be hidden inside when the protein is folded and, therefore, inaccessible to antibodies. Moreover, the sequences of the amino acids are different, meaning if this part of the protein were on the outside of the folded ball of protein, a scientist cannot assume that the immune response, taking the form of antibodies or T-cells, will be able to recognize it as its host sequence. Id. Dr. Whitton adds that even if this hypothetical cross-reactive immune response existed, a scientist would not assume it causes disease. Id. Dr. Whitton concludes that Dr. Steinman has not provided evidence that flu vaccine can cause any of the various diseases that have surfaced in his reports. Id. at 7.

Dr. Lancaster in his third supplemental expert report notes that the reported parts of petitioner's neurological examinations reflected in Ex. 207 were normal, weighing against a diagnosis of stiff person syndrome and not supportive of a diagnosis of active myasthenia gravis. Ex. QQQQ, at 1. Dr. Lancaster states that myasthenia gravis, autonomic failure, and dystonia are completely different disorders that affect different parts of the nervous system and have different causes. No one should conflate these diseases or refer to them interchangeably or consider them part of the same disease process. He also says that none of these disorders would account for petitioner's most prominent symptoms during the weeks and months after her flu vaccination. He also cautions we should consider petitioner's rapid responses to Dr. Buttar's treatment of these prominent symptoms in assessing the validity of their diagnoses. Id.

Dr. Lancaster states myasthenia gravis is an autoimmune disease of the muscular junction where nerve cells communicate with voluntarily-controlled muscles. Id. In most cases of myasthenia gravis, autoantibodies to the acetylcholine receptor cause the disease, with smaller groups having other autoantibodies. Id. at 1-2. He says GAD65 antibodies do not cause myasthenia gravis and are not used clinically as a diagnostic test for myasthenia gravis. Id. at 2. Dr. Lancaster notes that GAD65 antibodies are relatively common in the general population, leading to patients with GAD65 antibodies who also have myasthenia gravis by chance. He comments that petitioner's test results in general are unsupportive of a diagnosis of myasthenia gravis. He advises weighing the negative AChrR and MuSK tests against a diagnosis of myasthenia gravis. Id.

Dr. Lancaster states patients with myasthenia gravis have severe weakness with attacks occurring over many hours to days and weeks. They do not and cannot maintain wild lurching gaits. They do not and cannot run 5K races in the middle of an attack. They do not speak with a foreign accent. He says myasthenia gravis does not cause confusion, mental symptoms, or numbness. Anyone who watched petitioner's videos saw these symptoms clearly. He notes these videos are extremely valuable in showing the viewers what was actually wrong with petitioner in the weeks following vaccination. Id.

Dr. Lancaster states that all of petitioner's tests seeking objective confirmation of her diagnosis of having myasthenia gravis were negative. He believes it very unlikely that petitioner ever had myasthenia gravis. In her first years after the 2009 flu vaccination, no neurologist suspected she had or diagnosed her with myasthenia gravis. Dr. Lancaster says he disagrees strongly with Dr. Steinman that myasthenia gravis caused petitioner's tremors and spasms. Myasthenia gravis tends to make patients weak and slow, not cause the wild lurching gait manifest on the videos. Patients with myasthenia gravis may show a slight limb tremor due to difficulty holding the limb up while undergoing a severe attack, but that is not at all like the hyperkinetic movement petitioner had on the videos. A person in myasthenic crisis who tried this would simply collapse from exhaustion. Dr. Lancaster states a person with myasthenia gravis can have a weak, quiet speech pattern, but myasthenia gravis would not cause a person to speak loudly and clearly with a British accent.

Dr. Lancaster continues with an analysis of petitioner's alleged diseases. He states that abnormal brain pathways for controlling motor movements cause dystonia, which is abnormal posture or muscle tone. Id. The autonomic nervous system does not cause dystonia. Dystonia is therefore not a form of dysautonomia. Myasthenia gravis, which originates in the brain, is not the cause of dystonia. The type of dystonic gait petitioner manifested where she could walk backward but not forward is generally considered to be either genetically caused or idiopathic, but not autoimmune. Dr. Lancaster thinks petitioner did not have dystonia but conversion disorder. He says dystonia does not cause attacks of weakness, foreign accent syndrome, mental confusion, numbness, or seizure-like events. Dystonia is not a good explanation for petitioner's symptoms. Id.

Dr. Lancaster then focuses on autonomic failure, which involves damage to the autonomic nervous system, which regulates blood pressure, heart rate, sweating, shivering, salivation, reactivity of pupils to light, urination, and mobility of the gastrointestinal tract. Id. at 2-3. Generally, no one has voluntary control of these functions. Id. at 3. The autonomic nervous system does not control voluntary movements such as walking, talking, thinking, sensation of touch, and seizure-like events. He says an autonomic disorder could not cause the complex abnormal movements petitioner showed in the videos. Patients with autonomic failure affecting their blood pressure control cannot run a 5K race. No autonomic disorder comes and goes over a period of seconds, as petitioner's symptoms rapidly improved and worsened in the videos. Dr. Lancaster considers a diagnosis of autonomic failure to explain petitioner's primary symptoms "terrible." Id.

Dr. Lancaster moves on to an analysis of the diagnosis of petitioner with autoimmune autonomic neuropathy. He describes this as an incredibly severe autonomic disorder with widespread failure of the autonomic nervous system. A person's pupils do not react to light, he or she cannot maintain his or her blood pressure, the heart rate may become fixed and invariant, his or her sweating is impaired, etc. Id. Dr. Lancaster says petitioner did not have autoimmune autonomic neuropathy. He states a patient with autoimmune autonomic neuropathy would not run, would not walk with a lurching gait, and would not speak in one-word answers since the disease is confined to the autonomic parts of the peripheral nervous system. Id.

Dr. Lancaster then focuses on GAD65 antibodies, noting they are associated with several different disorders. He is unclear which of these disorders Dr. Steinman thinks petitioner had. Dr. Lancaster thinks it highly unlikely that petitioner had any of them. None of these disorders rapidly turns on and off over a period of seconds. Rather, they persist for many weeks to months and years. A patient with stiff person syndrome could not lurch and bob wildly but would have a slow, cautious, stiff gait. Someone with the incoordination of cerebellar ataxia who attempted the gait petitioner manifested would fall and injure himself or herself. Cerebellar degeneration causes a severe and persistent, often permanent, inability to control muscles. Patients with cerebellar degeneration are at severe risk of falling even when walking as normally as possible, and often need walkers even if they can walk. Id.

Dr. Lancaster states that Dr. Steinman's clinical diagnoses in his latest report are highly unlikely to be correct. Even if they were correct, they would not explain petitioner's course after her flu vaccination. Dr. Lancaster says the only reasonable diagnosis that explains petitioner's clinical findings is conversion disorder. GAD65 antibodies do not provide a rational explanation for her symptoms. One to two percent of the general population have GAD65 antibodies, especially at low titers. They are found in IVIG, raising the possibility that their presence in petitioner is an artifact of IVIG treatment. By low titers, Dr. Lancaster means up to 10-20 times normal. Id. In his practice, Dr. Lancaster evaluates GAD65 responses in spinal fluid in his patients. Id. at 3-4.

Dr. Lancaster says it is important to note that expert neurologists at Johns Hopkins evaluated petitioner when she was talking in one-word answers. Id. at 4. They did not diagnose petitioner with myasthenia gravis and she did not have myasthenia gravis. That diagnosis arose years after her vaccination. He reiterates that Dr. Steinman conflated two unrelated diseases, autoimmune autonomic neuropathy (involving peripheral nerves that control heart rate, blood pressure, sweating, pupillary constriction, and other autonomic functions) with dystonia (involving abnormalities in the tone and movement of muscles that are under voluntary control in the arms, legs, neck face, and voice) arising from abnormal motor control in the brain and not the autonomic nervous system. Id.

Dr. Lancaster says petitioner's GAD65 antibody test results have titers that fit his definition of a weakly positive GAD65 response. For example, a response of 6IU/ml with a normal range of 0-5IU/ml is only minimally positive. Other titer results were still less than 10 times the upper limit of normal, which would be 50 (petitioner's highest titer was 37IU/ml). Since petitioner's treatment included IVIG and GAD65 is commonly present in some of the

general population, she could possibly have this weak GAD65 result from her various doses of IVIG. In addition, Dr. Lancaster says we do not know when petitioner acquired these GAD65 antibodies since doctors first measured them years after her flu vaccination and onset of symptoms. Id. He notes petitioner does not have any of the neurological symptoms associated with GAD65 antibodies. Id. at 4-5.

Dr. Lancaster mentions that Dr. Gee's diagnosis of petitioner with stiff person syndrome is incorrect and was made years after her flu vaccination. Id. at 5. Dr. Lancaster states he treats stiff person syndrome in his clinic and he did not observe any signs of it in petitioner during the hearing. Not only does petitioner not have the symptoms of stiff person syndrome, but also the diagnosis would not explain her symptoms after her flu vaccination. A patient with stiff person syndrome does not have a wild lurching gait and, if he or she attempted such a gait, he or she would probably fall down. Stiff person syndrome does not cause unexplained weakness, foreign accent syndrome, seizure-like events, periods of apparent paralysis, numbness, and tongue paralysis. He notes stiff person syndrome would not come and go rapidly over seconds. Patients with stiff person syndrome do not walk better backward than forward. Instead, they would have more difficulty walking backward than walking forward. Dr. Lancaster concludes that none of petitioner's treating physicians, with good reason, in the years after the onset of her symptoms diagnosed her with stiff person syndrome. Id.

Dr. Lancaster also disagrees with Dr. Gee's diagnosis of petitioner having POTS. The diagnosis was made years after her vaccination, is mostly likely incorrect, and would not explain petitioner's symptoms in the month after flu vaccination. POTS was considered as a possible diagnosis in 2010. POTS manifests in lightheadedness, palpitations or something similar when standing, accompanied by an abnormal increase in pulse rate. POTS is a syndrome and not a disease, meaning the symptoms may have many causes. The possible causes could be deconditioning, dehydration, and prolonged bedrest. Dr. Lancaster says POTS is generally not an autoimmune disease. Id.

Dr. Lancaster notes that POTS is sometimes considered a disorder of the autonomic nervous system as a form of dysautonomia. Dr. Lancaster believes that petitioner did not have an autonomic disorder in the months after vaccination and he does not think she has POTS and that POTS is an explanation for her symptoms. Id. POTS would not cause foreign accent syndrome, wild lurching gait, the ability to run but not walk, abnormal postures, numbness, or symptoms occurring when supine. Id. at 5-6.

Dr. Lancaster thinks Mestinon at MedStar Georgetown University Hospital had a placebo effect on petitioner which is a reasonable possibility explaining her rapid recovery. Id. at 6. The videos with Dr. Buttar and petitioner show a very powerful placebo effect when Dr. Buttar rubbed chelation drops on petitioner's forearms. In response to Dr. Steinman's skepticism that a person could have GAD65 antibodies without also having neurological symptoms, Dr. Lancaster states it is far more likely that an individual has incidental GAD65 antibodies than the person has stiff person syndrome. Id. Dr. Lancaster states that in his practice, a high-titer GAD65 is several orders of magnitude above normal. Since normal is 0-5IU/ml, a strong positive titer would be >250IU/ml. Petitioner's GAD65 titer was as low as 6IU/ml and could not have been any lower

and be positive at all. Her strongest titer (37IU/ml) was less than 8 times normal, i.e., 40IU/ml. Id. Dr. Lancaster notes that Dr. Steinman neither endorses the diagnosis of stiff person disease in petitioner nor rejects it. How then can Dr. Steinman be providing evidence of petitioner having this disease if he is not willing to endorse it?

Dr. Lancaster states Dr. Steinman is skeptical that IVIG could have a placebo effect on petitioner's symptoms. Dr. Lancaster refutes that skepticism by noting IVIG has a well-recognized placebo effect. Id.

Dr. Lancaster disagrees with Dr. Steinman about the importance of the video evidence. Id. The central issue in this case is what occurred in the weeks and months after petitioner received flu vaccine. Id. at 7. Dr. Lancaster states the videos provide hours of objective evidence of what her symptoms and signs were. Id. Dr. Lancaster notes that Dr. Steinman was "extremely reticent" in stating which of petitioner's symptoms seen in the videos were due to which disease process. Dr. Lancaster says this is "the core of the case." Id. Dr. Lancaster notes that diagnoses proposed years later are too remote in time to illuminate what was happening contemporaneously with petitioner's vaccination in 2009. Id.

Dr. Lancaster questions the plausibility for petitioner's having all the illnesses with which doctors diagnosed her later on. He questions where petitioner's abnormal brain MRIs and abnormal CSF from her purported autoimmune encephalopathy are. He queries what specific type of autoimmune encephalopathy did petitioner purportedly have. He questions where the objective laboratory and electrophysiologic findings supporting a diagnosis of myasthenia gravis are. He queries what objective evidence exists for petitioner's having dysautonomia. He questions what type of autoimmune dystonia petitioner supposedly had. He notes that Dr. Steinman has not identified which type of dystonic disorder petitioner purportedly had or how flu vaccine could trigger it and why the immune system would be involved. Id.

Dr. Lancaster says if petitioner had myelin autoimmunity, she would be expected to have widespread and obvious lesions throughout her nervous system, not dystonia. Id. at 8. Dr. Lancaster states the four diagnoses Dr. Steinman proposes, i.e., autoimmune dysautonomia, seronegative myasthenia gravis, dystonia, and GAD65 (stiff person syndrome and/or cerebellar ataxia), are four completely different diseases affecting completely different parts of the nervous system. Id. Dr. Lancaster says none of the four diseases captures what happened to petitioner after flu vaccination, including cognitive difficulties, foreign accent syndrome, odd burning sensations, ability to walk backward or run but not walk forward, numbness, and seizure-like events. Id.

Medical Literature

Petitioner filed as reference 1 to Dr. Steinman's first expert report (Ex. 65) a case report by Sophie C. Skellett & Rosepal Dhesi, Myositis, rhabdomyolysis and compartment syndrome complicating influenza A in a child, BMJ CASE REP (Dec. 17, 2009), published online: doi:10.1136/bcr.07.2009.2099, as Exhibit 67. An eight-year-old boy with influenza A virus (not vaccine) came down with fever, lethargy, vomiting, and, on day four, myositis, rhabdomyolysis, renal failure, and compartment syndrome, leading to death from cardiorespiratory arrest and

multiorgan failure. The authors state the most common cause of viral myositis in children is influenza A and B. Id. at 3. In the case of the little boy, influenza A caused myositis, the muscle cells swelled, and he had compartment syndrome from elevated pressure in a closed fascial space. The authors note there are few case reports of rhabdomyolysis, renal failure, and compartment syndrome in adults. Id. at 4. They also note that viral myositis is uncommon in adults. Id. The authors also note that there are no reports of rhabdomyolysis, renal failure, and compartment syndrome associated with influenza infection in children who received flu vaccine. Id. at 5.

Petitioner filed as reference 2 to Dr. Steinman's first expert report (Ex. 65) a case report by Rodrigo B. Callado et al., Rhabdomyolysis Secondary to influenza A H1N1 Vaccine Resulting in Acute Kidney Injury, 11 TRAVEL MED & INFECTIOUS DIS 130 (2013), published online: https://doi.org/10.1016/j.tmaid.2012.11.004, as Exhibit 68. A 58-year-old man on statins developed rhabdomyolysis one day after receiving influenza A H_1N_1 vaccine. He went to the ED four days later and underwent hemodialysis to recover renal function. The authors note that one of the many factors causing rhabdomyolysis is exertion. Muscle toxicity has also been described as one of the major adverse effects of statins. The patient also had diabetes mellitus, hypercholesterolemia, coronary artery disease, and recurrent atrial flutter. The authors state that, although influenza B virus has been reported as the most common viral cause of myositis, influenza type A is associated more with rhabdomyolysis. Id. at 1. The patient had progressive symmetric quadriparesis, pain in the upper limbs and lower back, dark urine and dysuria. The patient had acute kidney injury classified as failure stage.

Petitioner filed as reference 6 to Dr. Steinman's first expert report (Ex. 65) a review by Haruki Koike et al., The Spectrum of Immune-Mediated Autonomic Neuropathies: Insights from the Clinicopathological Features, 84 J NEUROL NEUROSURG PSYCHIATRY 98 (2013), as Exhibit 72. The authors state that upper respiratory tract infections and gastrointestinal tract infections, which are likely due to viral infections, are the most common antecedent infections causing autoimmune autonomic ganglionopathies. Id. at 99. Case reports also indicate as causes preceding influenza A, infectious mononucleosis, mumps, epididymitis, aseptic meningitis, encephalitis, vaccination, surgical procedures, and interferon therapy. Id.

Petitioner filed as reference 16 to Dr. Steinman's first expert report (Ex. 65) an article by Yu-Zhong Wang et al., Expression of Toll-Like Receptors 2, 4, and 9 in Patients with Guillain-Barré Syndrome, 19 NEUROIMMUNOMODULATION 60 (2012), as Exhibit 82. The authors state that GBS is characterized by the progressive weakness of extremities, which mostly an antecedent respiratory or gastrointestinal tract infection triggers. Ex. 82, at 98.

Petitioner filed as reference 19 to Dr. Steinman's first expert report (Ex. 65) an article by Steven Vernino et al., Invited Article: Autonomic Ganglia. Target and Therapeutic Tool, 70 NEUR 1926 (2008), as Exhibit 85. The authors state that, in many cases, an antecedent viral syndrome such as upper respiratory symptoms or gastroenteritis precedes autoimmune autonomic ganglionopathy ("AAG"), but no specific infectious agent has been consistently identified. Id. at 1927. The authors note that about 14% of postural tachycardia syndrome

("POTS") cases have a subacute onset and may follow a viral prodrome, much as in the case of AAG. Id. at 1929.

Petitioner filed as reference 20 to Dr. Steinman's first expert report (Ex. 65) an article by Steven Vernino et al., Autoantibodies to Ganglionic Acetylcholine Receptors in Autoimmune Autonomic Neuropathies, 343 NEJM 847 (2000), as Exhibit 86. The authors state that clinical features of autoimmune autonomic neuropathies are: a subacute onset, prominent symptoms of gastrointestinal dysmotility, and an abnormal pupillary response to light and to accommodation. Ex. 86, at 851. Besides testing for gastrointestinal dysmotility, doctors would have to test for cardiovascular and sudomotor autonomic functions to diagnose autonomic neuropathy. Id. at 852. The authors note that autoimmune autonomic neuropathy is often a monophasic illness. Id. at 854. In September-December 2009, petitioner did not have gastrointestinal dysmotility or cardiac dysfunction and no one tested her for sudomotor autonomic function

Petitioner filed as reference 21 to Dr. Steinman's first expert report (Ex. 65) an article by Steven Vernino et al., Autonomic Ganglia, Acetylcholine Receptor Antibodies, and Autoimmune Ganglionopathy, 146 AUTON NEUROSCI 3 (2009), as Exhibit 87. The authors state that patients with autoimmune autonomic ganglionopathy ("AAG") typically do not have weakness or other clinical features of myasthenia gravis ("MG"). Id. at 6.

Respondent filed as reference 2 to Dr. Donofrio's expert report (Ex. B) an article by Walker B. Plash et al., Diagnosing Postural Tachycardia Syndrome: Comparison of Tilt Testing Compared with Standing Haemodynamics, 123 CLIN SCI 109 (2013), as Exhibit E. The authors disagree with the standard tilt-table test for POTS which is an increase in heart rate of 30 beats per minute within 10 minutes of assuming upright posture because active standing forces blood to return to the heart and results in passive tilt-test (false positive) results in nearly 15 percent of healthy subjects who experience vasovagal episodes. Ex. E, at 109. They note that a 10-minute tilt and a 30-minute tilt are not nearly as accurate when using 30 beats per minute as a cut-off. Id. at 112. The 10-minute tilt-test with a 30 beat per minute cut-off was highly sensitive but had poor specificity because it identified 60 percent of the control subjects as having POTS. Id. at 113. By increasing the heart rate cut-off to 37 beats per minute, the authors found the test was much more specific to those who had POTS while maintaining sensitivity. Id. The authors found that increasing the heart rate cut-off to 47 beats per minute increased the specificity to 80 percent while maintaining similar sensitivity. They note that an increase in heart rate is not the only criterion for diagnosing POTS. Patients must also have symptoms of presyncope or orthostatic intolerance. Id.

Petitioner filed as reference 3 to Dr. Steinman's first supplemental expert report (Ex. 92) an article by Blair P. Grubb et al., The Postural Orthostatic Tachycardia Syndrome: A Neurocardiogenic Variant Identified During Head-Up Tilt Table Testing, 20 PACE 2205 (1997), as Exhibit 97. In half of their POTS patients, a viral illness appeared to precede the onset of symptoms. Id. at 2210.

Petitioner filed as reference 1 to Dr. Steinman's second supplemental expert report (Ex. 108), a Medscape article by Michael T. Andary, Guillain-Barre Syndrome Clinical Presentation,

MEDSCAPE (Sept. 4, 2014), https://emedicine.medscape.com/article/315632-clinical-overview, as Exhibit 111. The author states that variants of GBS may present as acute dysautonomia. Ex. 111, at 1. The mean time to clinical function nadir is 12 days with 98% of patients reaching a nadir by four weeks. Then, they experience a plateau of persistent, unchanging symptoms, which gradual symptom improvement follows. Recovery usually begins two to four weeks after the progression of symptoms ends. Id. The author states dysautonomia is more frequent in patients with severe weakness and respiratory failure. It is important to note that petitioner's severe weakness was a consequence of rhabdomyolysis, not GBS, and she never had respiratory failure. The author of the Medscape article notes that up to two-thirds of GBS patients have an antecedent illness, usually upper respiratory and gastrointestinal, one to three weeks prior to onset of weakness. Id. at 2. The author states reflexes in GBS patients are absent or reduced early in the disease course. Id. at 3. Petitioner had normal reflexes.

Petitioner filed as reference 3 to Dr. Steinman's second supplemental expert report (Ex. 108) an article by Udaya Seneviratne (a neurologist at General Hospital, Ratnapura, Sri Lanka), Review. Guillain-Barré Syndrome, 76 POSTGRAD MED J 774 (2000), as Exhibit 110. Dr. Seneviratne is the sole author. She says autonomic dysfunction occurs in about two-thirds of GBS cases. Ex. 110, at 776. She also states that a variant of GBS includes pure dystonia.[146] Id. at 778. She states rarely autonomic neuropathy may be the presenting feature of GBS. Id. at 779. However, she also states that in order to diagnose GBS, progressive motor weakness and areflexia are prime requirements and CSF analysis is the only laboratory criterion. Id. at 780. Petitioner did not satisfy those prime requirements of GBS as she did not have motor weakness and areflexia and her CSF protein on lumbar puncture was normal.

[146] Dr. Seneviratne relies on two articles for her statement that a variant of GBS includes pure dystonia. The first is by Emilia-Romagna Study Group on Clinical and Epidemiological Problems in Neurology, Guillain-Barré Syndrome Variants in Emilia-Romagna, Italy, 1992-3: Incidence, Clinical Features, and Prognosis, 65 J NEUROL NEUROSURG PSYCHIATRY 218 (1998). The authors feature 11 patients. Id. at 220. None of these patients had pure dystonia. Six of them had either facial diplegia or bifacial weakness among other symptoms. One had asymmetric motor defect, nystagmus, and areflexia, among other symptoms. Another had upper limb motor defect with cervical pain. Another had pure sensory defect, absent ankle reflexes, and elevated CSF protein. The remaining patient had motor-sensory defect, abdominal hypesthesia, and sensory ataxia. Id. at 220-21. None of them had pure dystonia. The Emilia-Romagna Study Group on Clinical and Epidemiological Problems in Neurology does state a variant of GBS is pure pandysautonomia, citing to the same article Dr. Seneviratne cites as her second basis for writing pure dystonia can be a GBS variant. That article is the Ropper article. It does not however describe pandysautonomia or pure dystonia as a GBS variant. Allan H. Ropper, Unusual Clinical Variants and Signs in Guillain-Barré Syndrome, 43 ARCH NEUROL 1150 (1986). Dr. Ropper describes three patients with blurred or double vision, ptosis, marked oropharyngeal, neck, and shoulder weakness, respiratory failure with areflexia in the arms of two of the patients. Another had areflexia and elevated protein. The third had severe oropharyngeal weakness, ptosis, absent or low reflexes and an elevated CSF protein. The doctors considered whether these patients had botulism. Id. at 1150-51. Petitioner did not have any of these symptoms or signs. Dr. Ropper describes another three patients who had areflexic paraparesis resembling a spinal cord lesion with elevated CSF protein. Petitioner did not have any of these symptoms or signs. Id. at 1151. Dr. Ropper describes eight patients with severe ptosis and mild facial weakness early in their illness, resembling myasthenia gravis. Five of them developed typical GBS symptoms and eight developed the restricted pharyngeal-cervical-brachial form. Id. at 1151-52. Petitioner did not have any of these symptoms or signs. Dr. Ropper describes two patients with acute severe midline back pain followed by a typical generalized GBS, including bifacial paresis. Id. at 1152. Petitioner did not have any of these symptoms or signs.

Respondent filed as reference 1 to Dr. Lancaster's expert report (Ex. H) a review by Rodi Zutt et al., Rhabdomyolysis: Review of the Literature, 24 NEUROMUSCUL DISORD 651 (2014), as Exhibit J. The authors define rhabdomyolysis as the rapid breakdown of skeletal muscle fibers, which leads to leakage of potentially toxic cellular contents into systemic circulation. Id. at 651. About 26,000 cases of rhabdomyolysis are reported annually in the United States. Id. at 652. The authors state many symptoms of rhabdomyolysis are non-specific, including myalgia, swelling, and weakness. Id. Patients may have fever, nausea, emesis, confusion, agitation, delirium, and anuria. Id. Muscle weakness can occur in any part of the body, but most frequently occurs in proximal leg muscles. Id. Among numerous causes of rhabdomyolysis that the authors list are viral infections and extreme physical exertion. Id. at 254. If a patient experiences a first episode of rhabdomyolysis but no family history of it, the authors state an environmental factor is the most likely cause in 75 percent of cases. Id. at 255.

Respondent filed as reference 2 to Dr. Lancaster's expert report (Ex. H) a case report by Sneh V. Shah & Krishna Reddy, Rhabdomyolysis with Acute Renal Failure Triggered by the Seasonal Flu Vaccination in a Patient Taking Simvastatin, BMJ CASE REPORTS, published online: https://doi:10.1136.bcr.11.2009.2485 (2010), as Exhibit K. Noting that their case was extremely rare, the authors describe a man in his 70s taking a statin (simvastatin) who had symptoms of rhabdomyolysis within 24 hours of receiving seasonal flu vaccine. Id. at 1. He entered the hospital where he had hospital-acquired pneumonia and multiorgan failure. Id. The authors note that the patient did not have a history of any symptoms suggestive of an infectious illness before his vaccination and, therefore, the authors felt that viral infection was unlikely to be the cause of his rhabdomyolysis. Id. at 2. Because acute viral infections can cause rhabdomyolysis, the authors considered the patient's flu vaccination as the trigger of his rhabdomyolysis in the context of the risk factor of his taking a statin. Id.

Respondent filed as reference 4 to Dr. Lancaster's expert report (Ex. H) an article by Sonia Berrih-Aknin et al., Diagnostic and Clinical Classification of Autoimmune Myasthenia Gravis, 48-49 J AUTOIMMUN 143 (Feb – Mar 2014), published online: https://doi.org/10.1016/j.jaut.2014.01.003, as Exhibit M. In intermediate cases of myasthenia gravis, symptoms include marked fatigue, impaired swallowing, a hypernasal voice, and diplopia. Id. at 143. Severe myasthenia gravis involves respiratory muscles and severe swallowing disorders. Id. They note a myasthenia crisis could be life-threatening involving shortness of breath, choking, and rapid motor deterioration. Id. at 146. They say the number of myasthenia patients with unknown antibodies is less than 5 percent. Id. at 147.

Respondent filed as reference 5 to Dr. Lancaster's expert report (Ex. H) an article by Eduardo E. Benarroch, The Clinical Approach to Autonomic Failure in Neurological Disorders, 10 NAT REV NEUROL 396 (2014) as Exhibit N. Benarroch states, "Many symptoms attributed to 'dysautonomia' in otherwise healthy young patients, such as gastroparesis or urinary retention, are rarely associated with objective evidence of autonomic failure. Id. at 396. He also states that onset of symptoms of autoimmune autonomic ganglionopathy may follow a viral infection, minor surgical procedure, or vaccination. Id. at 401. He mentions adverse effects for drugs prescribed for orthostatic hypotension: (1) midodrine - scalp tingling; (2) pyridostigmine -

nausea, abdominal cramps, and diarrhea; (3) octreotide - nausea and abdominal cramps. Id. at 403. Petitioner was taking these drugs.

Respondent filed as reference 4 to Dr. Whitton's expert report (Ex. Z) a case report by Michael D. Nauss et al., Viral Myositis Leading to Rhabdomyolysis: A Case Report and Literature Review, 27 AM J EMERG MED 372.e5 (2009), as Exhibit EE. The authors state, "Several viruses have been implicated in rhabdomyolysis: influenza A/B, parainfluenza, coxsackie, Epstein-Barr, herpes simplex, adenovirus, and cytomegalovirus." Id. at 372.e5. In their case report, a 29-year-old man came to the ED, complaining of dark urine and muscle pain. About 10 days before, he had developed fevers and a cough. Four days before going to the hospital, he developed muscle swelling in his calves and arms associated with weakness. The authors note that, in adults, the most common causes of rhabdomyolysis are exertion, crush injury, seizures, alcohol, viruses, drug abuse, and use of statins. Id. They posit three possible causes to explain viral-associated myositis: (1) direct viral invasion of myocytes; (2) viral mediated myotoxic cytokines released by infection; these cytokines include tumor necrosis factor that can cause skeletal muscle breakdown; and (3) autoimmune myositis resulting from the viral infection. Id. at 372.e6. The time from viral illness to the onset of myositis varies from coincidental to a three-week delay. Id.

Respondent filed as reference 6 to Dr. Whitton's expert report (Ex. Z) a case report by Nikhil Sharma et al., Exercise-Induced Rhabdomyolysis: Even the Fit May Suffer, 53 INT J CLIN PRACT 476 (1999), as Exhibit GG. The authors report the case of a female 29-year-old family doctor who had been using a gym two or three times a week for about four years, exercising for 60 minutes including resistance work followed by a 15-minute swim. Id. at 476. Three days before she came to the hospital, she attempted a new exercise called body biking which entailed standing on the pedals of a stationary bike for 40 minutes, moving up and down against resistance, followed by a swim of 10 minutes. The next day, her thighs were excessively painful and slightly swollen. The pain worsened considerably 24 hours later and her urine turned dark brown. She was on oral contraceptives but took no other medication. The hospital diagnosed her with rhabdomyolysis. The authors state an increase in intracellular calcium is an important factor in the muscle injury. Damage to muscle cell membranes leads to an influx of sodium which disrupts the relationship between intracellular sodium and calcium, leading to a rise in intracellular calcium. Id. The authors say the development of rhabdomyolysis following exercise depends on the extent of the muscular activity, the condition of the athlete, and the type of muscular contraction involved. Id. at 477. They conclude, "Even a young, fit woman without apparent risk factors and who exercises regularly can develop acute rhabdomyolysis with only a modest change to her exercise routine." Id.

Respondent filed as reference 7 to Dr. Whitton's expert report (Ex. Z) an article by Pieter A. van Doorn et al., Clinical Features, Pathogenesis, and Treatment of Guillain-Barré Syndrome, 7 LANCET NEUROL 939 (2008), as Exhibit HH. The authors describe the first symptoms as pain, numbness, paresthesia, or weakness in the limbs. Ex. HH, at 939. The main features of GBS are rapidly progressive bilateral and relatively symmetric weakness of the limbs with or without involvement of respiratory muscles or cranial-nerve-innervated muscles. Id. CSF examination

by lumbar puncture typically shows increased protein. Patients have decreased or absent deep tendon reflexes. Id. Citing a Japanese study, the authors state the most frequent antecedent symptoms in GBS were fever, cough, sore throat, nasal discharge, and diarrhea. Id. at 940. They state, "an argument for the post-infectious nature of GBS is the typical monophasic clinical course of the disease." Id.

Petitioner filed as reference 7 to Dr. Steinman's third supplemental expert report (Ex. 118) an article by Arthur K. Asbury et al., The Inflammatory Lesions in Idiopathic Polyneuritis. Its Role in Pathogenesis, 48 MEDICINE 173 (1969), as Exhibit 126. The authors studied 19 cases of people with polyneuritis who died. They state the clinical picture encountered most frequently was weakness progressing to paralysis in legs, arms, and respiratory musculature over three to seven days. Ex. 126, at 201. Motor weakness overshadowed sensory disturbances in all cases, but most patients complained of paresthesia and tingling. Id. As the illness evolved, CSF protein levels rose. Id. at 202. The pathologic hallmark of idiopathic polyneuritis is perivenular mononuclear inflammatory infiltrate, which occurred in all 19 cases. Id. They found lesions throughout the peripheral nervous system. Id. at 203. Petitioner did not have any lesions in her peripheral nervous system as all her EMGs and nerve conduction studies were normal.

Petitioner filed as reference 10 to Dr. Steinman's third supplemental expert report (Ex. 118) an article by Stanley Fahn, Clinical Variants of Idiopathic Dystonia, J NEUR NEUROSURG & PSYCHIATRY Supp 96 (1989), as Exhibit 129. Fahn states, "The presence of clinical clues, such as fake weakness, somatisations [sic], and deliberate slowness of movement, serve to lead one to the diagnosis of psychogenic dystonia." Id. at 97.

Petitioner filed as reference 11 to Dr. Steinman's third supplemental expert report (Ex. 118) an article by Alexander G. Munts & Peter J. Koehler, Occasional Paper. How psychogenic is dystonia? Views from Past to Present, 133 BRAIN 1552 (2010), as Exhibit 130. The authors list seven reasons why focal dystonias have been regarded as psychogenic: (1) the bizarre nature of the dyskinesias; (2) their appearance frequently only during certain actions while other motor acts using the same muscles are done normally; (3) certain inexplicable trick actions relieving the dyskinesias; (4) the manifestation of dyskinesias being exquisitely sensitive to social and mental stress; (5) the failure to find any anatomic, physiologic, or biochemical abnormality in any of the dyskinesias; (6) the impression that the patient shows overt psychiatric disturbance; and (7) a psychopathologic interpretation of the significance of dyskinesias such as eye closure or neck turning. Ex. 130, at 9.

Petitioner filed as reference 13 to Dr. Steinman's third supplemental expert report (Ex. 118) an article by Guillermo A. Suarez et al., Idiopathic Autonomic neuropathy: Clinical, Neurophysiologic, and Follow-up studies on 27 Patients, 44 NEUROLOGY 1675 (1994), as Exhibit 132. The authors included from their study those who had neurotoxic drug exposure and those with acquired polyneuropathies, i.e., chronic inflammatory demyelinating polyneuropathy ("CIDP"), acute inflammatory demyelinating polyneuropathy ("AIDP"), and neuropathies associated with monoclonal gammopathy of undetermined significance. Ex. 132, at 1. Dr. Steinman uses the Suarez article as proof for his thesis that someone with GBS or AIDP can have a primary autonomic nervous system disease, i.e., that patients with idiopathic autonomic

neuropathy are on a spectrum with patients who have AIDP, yet the Suarez authors explicitly eliminated patients with AIDP from their study. The authors also excluded patients with POTS. Id. Petitioner allegedly has POTS, which means the authors would have excluded her from this study. The authors also excluded patients on anticholinergic and antidepressant medications, id., which would have been a further reason to exclude petitioner from their study.

The authors thoroughly tested the patients with various tests that would prove autonomic failure, including the thermoregulatory sweat test, the quantitative sudomotor axon reflex test (QSART), abnormal EMG, and plasma norepinephrine testing in both supine and upright positions. Id. at 2. Petitioner either passed these tests or never took them. The authors again would not have included her in patients with idiopathic autonomic neuropathy.

Twenty-seven patients, including 18 females and nine males, met the authors' criteria for idiopathic autonomic neuropathy. Id. at 3. Their ages ranged from 7 to 75 years, with a mean of 45 years. The onset of symptoms was acute (less than two weeks) in 10 patients, subacute (up to eight weeks) in 12 patients, and gradual in five. Id. In 16 patients, a presumed viral infection preceded the onset of symptoms. An antecedent or concurrent flulike syndrome or upper respiratory infection occurred in 11 patients. Id.

The most common symptoms, occurring in 21 out of 27 patients, were orthostatic symptoms, e.g., persistent and severe lightheadedness, dizziness, or near syncope upon standing. Nineteen patients reported gastrointestinal symptoms, e.g., nausea, vomiting, diarrhea, constipation, and postprandial bloating. Seventeen patients had symptoms of thermoregulatory impairment and heat intolerance, e.g., becoming hot, dizzy, and flushed during exercise or elevated temperatures but would not sweat. Ten patients had visual and ocular symptoms, e.g., blurred vision, photophobia, and dry eyes. Nine patients had urinary symptoms, e.g., difficulty voiding to urinary retention. Seven patients (26%) had neuropathic symptoms to tingling and numbness in their feet and hands. Id.

Except for variable postural hypotension, 14 patients had normal neurologic exams. Six patients had mild limb weakness. Nine patients had impaired deep tendon reflexes. Seven patients had distal sensory deficits. The CSF protein level in 10 of 27 patients was evaluated with a mean slight elevation of 46.9mg/dL with a range from 22 to 67mg/dL. No other serologic or immunologic abnormalities were recorded in lab studies. Id.

The authors surmised that the majority of these 27 patients had a monophasic course with a progressive phase followed by a plateau and remission, or a prolonged stable deficit without remission or recurrence. Id. at 6. There was a trend to recovery of function in two and one-half years, but not complete recovery. Severe autonomic failure in 17 patients at initial presentation was the most common degree of abnormality (63%). Id.

The authors suspected the lesion is in the peripheral (preganglionic or postganglionic[147]) nervous system for the following reasons: (1) none of the 27 patients had clinical evidence of

[147] Preganglionic is "situated anterior or proximal to a ganglion; said especially of autonomic nerve fibers so located." Dorland's at 1509. Postganglionic is "situated posterior or distal to a ganglion; said especially of autonomic nerve fibers so located." Id. at1502. A ganglion is "anatomic terminology for a group of nerve cell

CNS involvement (brain/spinal cord); (2) 24 patients had evidence of postganglionic sudomotor denervation on the summated QSART (distal legs and proximal foot); (3) combining the results of the TST and the summated QSART, the authors note 20 patients had evidence of a peripheral postganglionic lesion; (4) three patients had electrophysiologic evidence of peripheral somatic nerve involvement on EMG; (5) four patients had histopathologic evidence of a neuropathic process on nerve biopsy; and (6) recovery of function, although incomplete, occurred in the majority of patients. Id. at 7. The authors state this recovery argued against a central lesion because central axons do not regenerate whereas peripheral axons can regenerate. Id.

The authors conclude that significant overlap of symptoms of idiopathic autonomic neuropathy with those who have AIDP or GBS exists since persons with AIDP or GBS can have autonomic involvement. Id. That is a different statement than Dr. Steinman makes, i.e., that someone with an autonomic nervous system disease has AIDP or GBS because someone with AIDP or GBS can have autonomic nervous system symptoms as well. The spectrum the authors create is not AIDP/GBS but of idiopathic autonomic neuropathy whose spectrum runs from idiopathic autonomic neuropathy to AIDP/GBS. They state, "Idiopathic autonomic neuropathy is at one end of the spectrum and AIDP is at the other, as the brunt of the disorder affects the somatic nervous system in AIDP." Id.

Of note, Dr. Steinman quotes in his expert report a sentence from the abstract of the Suarez article which is not verbatim in the article itself: "Pathologic features include the presence of a small inflammatory mononuclear cell infiltrate[148] in the epineurium.[149]" Cf. Ex. 118, at 21, with Ex. 132, at 1 (abstract). By including that sentence in his expert report, Dr. Steinman conveys the impression that this pathologic finding was common in the Suarez article's study of 27 patients with idiopathic autonomic neuropathy. This is not true. The authors found this pathologic presence in only one patient. Ex. 132, at 5. In a section entitled "Pathologic studies," the authors describe different pathologic results from different patients among the 27 who were the subject of the article. The authors state without comment, "A small mononuclear cell infiltrate surrounding an epineurial vessel, without changes typical of necrotizing vasculitis, was found in one patient." Id.

Petitioner filed as reference 14 to Dr. Steinman's third supplemental expert report (Ex. 118) an article by Joan Sneddon, Myasthenia Gravis—The Difficult Diagnosis, 136 BRIT J PSYCHIAT 92 (1980), as Exhibit 133. The author describes four cases of myasthenia gravis which had delayed diagnosis. She goes on to write:

> Pseudo-myasthenia gravis must also be mentioned [citation omitted]. Such patients, mainly women, show this syndrome as

bodies located outside the central nervous system." Id. at 757. "Anterior" means "in front of." Id. at 98. "Posterior" means "situated in back of." Id. at 1502.

[148] Infiltration is "the pathological accumulation in tissue or cells of substances not normal to it or in amounts in excess of the normal." Dorland's at 936. Cellular infiltration is "the migration and accumulation of cells within the tissues." Id. at 936. Mononuclear means "a cell having a single nucleus, especially a monocyte of the blood or tissues." Id. at 1177.

[149] The epineurium is "the outermost layers of connective tissue of a peripheral nerve, surrounding the entire nerve and containing its supplying blood vessels and lymphatics." Dorland's at 634.

> part of a long history of conversion hysteria. They have some of the symptoms of myasthenia and are able to tolerate large doses of cholinesterase inhibitors without getting side-effects but if the drugs are withdrawn do not go into myasthenic crisis.

Ex. 133, at 93.

Petitioner filed as reference 17 to Dr. Steinman's third supplemental expert report (Ex. 118) a review article by Glenis K. Scadding & C.W.H. Havard, Pathogenesis and Treatment of Myasthenia Gravis, 283 BMJ 1008 (1981), as Exhibit 136. The authors state that typically someone with myasthenia gravis has worse muscle weakness after effort which is improved by rest. Ex. 136, at 1008. Interestingly, petitioner felt better when she ran than when she was at rest. In 2011, she was exercising 20 hours a week, which is nearly three hours a day.

The authors note that generalized myasthenia has three clinical patterns: (1) association with thymoma;[150] (2) association with thymitis[151] under the age of 40; and (3) association with thymitis over the age of 40. Id. Petitioner does not fall into any of these three clinical patterns. She did not have thymoma or thymitis.

Petitioner filed as reference 19 to Dr. Steinman's third supplemental expert report (Ex. 118) a case report by Jacqueline I. Bakker et al., Foreign Accent Syndrome in a Patient with Multiple Sclerosis, 31 CAN J NEUROL SCI 271 (2004), as Exhibit 138. The authors report a 52-year-old woman with relapsing remitting MS who had a Dutch accent with other neurologic symptoms that resolved simultaneously. Ex. 138, at 271. Her brain MRI showed deep white matter lesions in the corpus callosum, left parietal lobe, and left frontal lobe, which the authors said were consistent with previous reports of foreign accent syndrome. Id. at 271-72. The patient recovered from foreign accent syndrome after she received treatment with high dose methylprednisolone. Id. at 272. The authors review reported cases of foreign accent syndrome, noting that most of the reported cases involve lesions in the dominant inferior dorsolateral premotor cortical-striatal-pallidal-thalamic circuit which mediates motor speech planning. Id. The authors opine the patient's MS caused her episodes of foreign accent. In the instant action, petitioner's brain MRIs were normal and she does not have MS.

Respondent filed as reference 1 to Dr. Lancaster's second supplemental expert report (Ex. RR) a review article by Donald B. Sanders & Jeffrey T. Guptill, Myasthenia Gravis and Lambert-Eaton Myasthenic Syndrome, 20 CONTINUUM 1413 (2014), as Exhibit WW. The authors state, "Diseases that affect the neuromuscular junction are characterized by weakness that predominantly affects certain muscle groups and fluctuates over time, worsening with use and improving after rest." Ex. WW, at 1413. In contrast, petitioner stated to her doctors on numerous occasions that she felt better exercising and worse at rest, which is the exact opposite of what a patient with myasthenia gravis should be experiencing.

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[150] Thymoma is "a tumor derived from the epithelial or lymphoid elements of the thymus." Dorland's at 1925. The thymus is "a bilaterally symmetric lymphoid organ consisting of two pyramidal lobes situated in the anterior superior mediastinum. … The thymus is the site of production of T lymphocytes." Id.

[151] Thymitis is "inflammation of the thymus." Dorland's at 1924.

Respondent filed as reference 5 to Dr. Lancaster's second supplemental expert report (Ex. RR) an article by Mark Hallett et al., Psychogenic Movement Disorders, 18S1 PARKINSONISM REL DISORD S155 (2012), as Exhibit ZZ. The authors list clues to diagnose a psychogenic movement disorder: (1) movements may be inconsistent over time; (2) tremors may come and go and vary in frequency; (3) movements are sometimes difficult to classify and may be a mixture of disorders, such as myoclonus, chorea, and dystonia; (4) movements might be so unusual that a doctor may view them as bizarre; (5) distraction may cause a movement to disappear; and (6) suggestion might precipitate a movement to appear. Ex. ZZ, at S156. Someone with a psychogenic movement disorder might have extreme slowness, sometimes appearing markedly fatigued. On physical examination, the patient might have give-way weakness or psychogenic patterns of sensory loss. Id. Psychogenic tremor is often highly variable in frequency, direction, and amplitude. The tremor can vary or cease with distraction. The authors write psychogenic gait is characterized by unusual patterns of stance and gait which are often inconsistent and dramatic with lurching but with only rare falls and no injury. Sudden knee buckling without falling is a common pattern. Id. Petitioner manifested all these characteristics of psychogenic movement.

Respondent filed as reference 1 to Dr. Whitton's third supplemental expert report (Ex. SS) an article by Bianca van den Berg et al., Guillain-Barré Syndrome: Pathogenesis, Diagnosis, Treatment and Prognosis, 10 NAT REV NEUROL 469 (2014), as Exhibit TT. The authors state, "GBS typically occurs after an infectious disease in which the immune response generates antibodies that cross-react with gangliosides at nerve membranes." Ex. TT, at 469. They note that GBS is characterized by rapidly progressive, symmetrical limb weakness with hyporeflexia or areflexia. Id. at 470. They also state progressive weakness reaches its maximum within four weeks and often within two weeks. Id. The authors state "vaccinations might even reduce the risk of acquiring GBS, as this condition can be caused by infections such as influenza." Id. at 472. They state the risk of developing GBS after flu infection is estimated to be four to seven times higher than after flu vaccination. Id.

Petitioner filed as reference 3 to Dr. Steinman's fourth supplemental report (Ex. 141) an article by Daniel B. Drachman, The Biology of Myasthenia Gravis, 4 ANN REV NEUROSCI 195 (1981), as Exhibit 144. The author states the cardinal features of myasthenia gravis consist of weakness and fatigue of skeletal muscles, involving impairment only of the motor system. Ex. 144, at 1-2. Sensation, reflexes, coordination, and other neural functions remain normal. Id. at 2. Often ptosis and diplopia are involved. "In severe cases, the patient's life may be endangered by weakness of the muscles of respiration and swallowing." Id. Muscle strength rapidly fatigues on repeated or sustained contraction and may improve after resting.

Petitioner filed as reference 5 to Dr. Steinman's fourth supplemental report (Ex. 141) an article by Irving Nachamkin et al., Anti-Ganglioside Antibody Induction by Swine (A/NJ/1976/H1N1) and Other Influenza Vaccines: Insights into Vaccine-Associated Guillain-Barré Syndrome, 198 J INFECT DIS 226 (2008), as Exhibit 146 (respondent also filed this as Ex. LLL before the hearing). The authors suspected that swine flu vaccine contained contamination from Campylobacter jejuni ("C. jejuni") antigens that mimic human gangliosides because more

GBS cases occurred after swine flu vaccination compared to the occurrence of GBS in the unvaccinated population. Ex. 146, at 227. They tested swine flu vaccine and flu vaccines manufactured for the 1991-1992 and 2004-2005 flu seasons in mice. Id. They did not detect anti-hemagglutinin antibodies on day zero but they detected significantly increased titers on days 21 and 35. Id. at 228. The authors discovered no contamination from C. jejuni, but all three vaccines, i.e., swine flu, and flu vaccine from 1991-1992 and 2004-2005, induced IgG and IgM antibodies to gangliosides even though the flu vaccines for 1991-1992 and 2004-2005 did not manifest an increased incidence of GBS among the vaccinated. Id. at 230. The authors speculate that low levels of viral neuraminidase ("NA") in the 1976 swine flu vaccine may have allowed sufficient sialic acid to remain bound to viral hemagglutinin ("HA") forming a sialic acid-HA complex that mimicked human ganglioside. Id. at 231. They state higher levels of viral NA in other flu vaccines could be sufficient to reduce the amount of sialic acid-HA complex so that the vaccine was less immunogenic and did not trigger GBS as swine flu vaccine did. They conclude that the immunogenicity of HA differs among flu viral strains and that was the reason that swine flu vaccine had different immunogenic properties than other flu vaccines, resulting in a more potent anti-ganglioside antibody response and GBS in susceptible recipients of swine flu vaccine compared to those receiving other flu vaccines. Id. This explains to the authors why "it has been difficult to establish a link between GBS and influenza vaccine-related GBS in recent years." Id. Dr. Steinman ignores in his report (Ex. 141, at 6) the conclusion of Nachamkin and his co-authors that only swine flu vaccine resulted in a greater incidence of GBS and subsequent flu vaccines have not resulted in a greater incidence of GBS than above baseline.

Petitioner filed as reference 11 to Dr. Steinman's fourth supplemental report (Ex. 141) an article by Silva Markovic-Plese et al., High Level of Cross-Reactivity in Influenza Virus Hemagglutinin-Specific CD4+ T-cell Response: Implications for the Initiation of Autoimmune Response in Multiple Sclerosis, 169 J IMMUNOL 31 (2005), as Exhibit 152. The authors focused on T-cell receptor ("TCR") cross-reactivity. Ex. 52, at 31. They recognize the pathogenic role of autoreactive T-lymphocytes in initiating an autoimmune response in MS has yet to be determined. Id. In order to study the requirements for molecular mimicry, the authors examined the flexibility of TCR recognition and the degree of sequence homology required for a cross-reactive immune response. Id. They identified a hierarchy of antigen specificities for a clone generated against immunodominant flu virus hemagglutinin ("Flu-HA") epitope within the setting of an acute viral infection in someone with MS. Id. at 32. They noted a high stimulatory potency of two MOG- and one CNPase-derived peptide as antigens capable of inducing cross-reactive responses in a particular CD4+ T-cell clone ("TCC"). Id. at 37. The authors discovered the CNPase-derived peptide had no homology with the native Flu-HA epitope and, therefore, major histocompatibility complex ("MHC") or TCR binding motif searches would not detect it. They concluded that molecular mimicry, although it occurs frequently, leads to autoimmune disease only in the context of chronic local inflammation, the presentation of self-antigens, and a sufficient number of autoreactive T-cells. Id. Dr. Steinman, at page 17 of his fourth supplemental report, uses the Markovic-Plese article to support his view that the 2009 flu vaccine caused a reaction to petitioner's myelin. He omits Markovic-Plese's conclusion that the CNPase-derived peptide had no homology to influenza virus A. Dr. Steinman also does not include Markovic-Plese's caveat that molecular mimicry would occur only in the context of

chronic local inflammation, the presentation of self-antigens, and a sufficient number of autoreactive T-cells.

Petitioner filed a 1974 article to which Dr. Steinman referred in his fourth supplemental report (Ex. 141) on pages 20-21, although he did not list it as a reference, by Charles David Marsden & M.J.G. Harrison, Idiopathic Torsion Dystonia (Dystonia Musculorum Deformans). A Review of Forty-Two Patients, 97 BRAIN 793 (1974), as Exhibit 170. The authors state it is frequently an inherited disease beginning in childhood and relentlessly progressive so that the patient is inevitably crippled by grotesque involuntary movements and postures by the time he or she becomes an adult. Ex. 170, at 1. However, they note that the illness can vary in severity, age of onset, and prognosis. Id. In order to participate in the authors' study, someone could not have evidence of cerebellar or sensory deficit on examination. Id. at 2. The authors studied 42 patients. The most common symptom was the difficulty in using one or both arms. Id. at 3. (Petitioner complained more about her legs than her arms.) The second most common feature was gait abnormality. Id. at 4. One-third of the patients were bed- or chair-bound. Id. at 6. Intelligence, personality and memory appeared unaffected in all 42 patients. Id. at 7. (Petitioner complained of cognitive impairment and decreased memory.) In their adult cases, only two patients (both younger than 21) had problems with gait disturbance. Id. at 9. (Petitioner's primary manifestation was gait disturbance.) The authors state adult onset dystonia has a relatively benign prognosis. Id. at 10.

Before the hearing, respondent filed an article by Ting Lei et al., Anti-Ganglioside Antibodies Were Not Detected in Human Subjects Infected with or Vaccinated Against 2009 Pandemic Influenza A (H1N1) Virus, 30 VACCINE 2605 (2012), as Exhibit OOO. The authors attempted to find anti-ganglioside antibodies in humans and mice vaccinated against 2009 pandemic H_1N_1 flu virus. Ex. OOO, at 2606. Eight of the persons had post-vaccination GBS, yet the authors did not detect anti-ganglioside antibodies in them or in the vaccinated mice. Id. at 2609. The authors also checked for anti-ganglioside antibodies in people infected with 2009 H_1N_1 virus and did not detect anti-ganglioside antibodies. Id. They state, "Our work did not support the model in which influenza vaccines induce anti-[ganglioside] antibodies in many recipients, which subsequently cause GBS in a small subset of individuals." Id. They also state that GBS associated with flu virus infection or flu vaccination might be pathologically different from GBS associated with an infection such as Campylobacter jejuni or another pathogen, in which anti-ganglioside antibodies were more frequently detected. Id.

On June 18, 2016, after the hearing held from June 14-17, 2016, petitioner filed additional articles marked during the hearing. One was by Satish R. Raj et al., Blood Volume Perturbations in the Postural Tachycardia Syndrome, 334 AM J MED SCI 57 (2007), as Exhibit 176. The authors state that patients with POTS have chronic symptoms lasting at least six months, consisting of rapid palpitation, exercise intolerance, lightheadedness, extreme fatigue, and mental clouding. Ex. 176, at 57. They have an increase in heart rate of at least 30 beats/min within 5 to 30 minutes of assuming an upright posture which should occur in the absence of orthostatic hypotension, i.e., a fall in blood pressure >20/10mm Hg. The authors also state many patients with POTS have low blood volume. Id. The authors theorize that abnormalities in the

renin-angiotensin-aldosterone axis may play a role in the pathophysiology of POTS by contributing to hypovolemia, perhaps by impaired sodium retention. Id. at 58. They trace perturbations in the renin-aldosterone system to partial sympathetic denervation involving the kidney, an explanation consistent with the partial dysautonomia hypothesis for some patients with POTS. Id. The recommendation to increase salt in the diet and intake of water is commonly made for POTS patients.

On October 14, 2016, petitioner filed case reports that Dr. Steinman listed as references 4, 5, and 6 in his sixth supplemental report (Ex. 191). The first reference is a case report by Yi-Ting Hsu et al., Polyglandular Autoimmune Syndrome Type 4 with GAD Antibody and Dystonia, 114 CLIN NEURO & NEUROSURG 1024 (2012), as Exhibit 194. A 20-year-old man had limb twisting for four years, type 1 diabetes mellitus, right hand and left leg twisting triggered by action or postural maintenance, alopecia and skin vitiligo, myasthenia gravis, and action-triggered left hand twisting movement. Ex. 194, at 1024. Polyglandular autoimmune syndrome ("PAS") is reported to be associated with various extrapyramidal disorders. Id. at 1026. The patient's brain MRI showed striatal necrosis. (Petitioner does not have polyglandular autoimmune syndrome or striatal necrosis of her brain.) The authors mention that nearly all patients with stiff person syndrome, cerebellar ataxia, epilepsy or myoclonus are affected by polyglandular autoimmune syndrome. (Petitioner does not have stiff person syndrome, cerebellar ataxia, epilepsy or myoclonus.) Interestingly, the authors state that their patient's dystonia was poorly responsive to immunotherapy, implying that his immune-mediated cause of dystonia was different than that of myasthenia gravis, vitiligo, or alopecia in PAS. Id.

The second reference is a case report by Jana Kenda et al., (Pseudo)hemidystonia Associated with Anti-Glutamic Acid Decarboxylase Antibodies – A Case Report, 22 EUR J NEUR 1573 (2015), as Exhibit 195. A 55-year-old woman with type 1 diabetes mellitus, chronic lymphocytic thyroiditis, and vitiligo presented with abnormal painful posturing of the left hemibody. She had a high titer of autoantibodies against GAD in both her CSF and serum. She was diagnosed with stiff person syndrome. Id. The authors mention pseudodystonia which is abnormal postures of body parts not caused by disorders of basal ganglia. Id. at 1574. Petitioner does not have any disorder of her basal ganglia.

The third reference is a "short report" by Angela Vincent et al., Antibodies to ^{125}I-glutamic acid decarboxylase in patients with stiff man syndrome, 62 J NEUR NEUROSUR & PSYCH 395 (1997), as Exhibit 196. The authors state stiff man syndrome involves muscle rigidity and cramps resulting from immune-mediated inhibition of GABAergic neuron function. Ex. 196, at 395. Some cases occur with type 1 diabetes mellitus and others are paraneoplastic, with breast tumors being most common. Anti-GAD antibodies are present in about 40 percent of patients with stiff man syndrome. Id. Of 15 patients with myasthenia gravis and 30 with acquired neuromyotonia, only one patient had clearly raised anti-GAD antibodies (this patient had neuromyotonia and a thymoma). Id. at 396-97. (Petitioner has not been diagnosed with neuromyotonia. She does not have a thymoma.) Interestingly, the authors found no anti-GAD antibodies among 15 patients with myasthenia gravis and only one patient out of 30 with acquired myotonia (and a thymoma) who had raised anti-GAD antibodies. Dr. Steinman

emphasizes in his fifth supplemental report (Ex. 191) how petitioner's positive anti-GAD antibodies are a basis for many of petitioner's problems. Ex. 191, at 10. Dr. Steinman does not specify what those many problems are, but this short report does not provide support for any correlation between anti-GAD antibodies and myasthenia gravis.

Dr. Steinman has an additional reference to a case report in his sixth supplemental expert report to emphasize that people with stiff man syndrome are frequently diagnosed as having a psychogenic movement disorder. Ex. 191, at 11. But that does not mean that someone who has a psychogenic movement disorder actually has stiff man syndrome. The case report petitioner filed is by E. Andreadou et al., Stiff Person Syndrome: Avoiding Misdiagnosis, 28 NEUROL SCI 35 (2007), as Exhibit 197. A 41-year-old woman had spasms and intense painless clonic jerks of her trunk and limbs lasting 30 minutes to two hours without impairment of consciousness. Ex. 197, at 35. She had profuse sweating and tachycardia. Id. Her EMG was abnormal, showing spontaneous involuntary normal motor unit potentials in lumbar paraspinal and abdominal muscles and in lower limbs in agonist and antagonist muscles simultaneously. Id. at 36. She had anti-GAD65 antibodies in her blood. Id. Petitioner never had an abnormal EMG.

Respondent filed as reference 4 to Dr. Whitton's third supplemental expert report (Ex. ZZZ) a report of two cases by Benjamin Lichtiger & Karen Rogge, Spurious Serologic Test Results in Patients Receiving Infusions of Intravenous Immune Gammaglobulin, 115 ARCH PATHOL LAB MED 467 (1991), as Exhibit DDDD. The authors state intravenous immune gammaglobulin is a concentrate of IgG coming from the plasma of a large number of donors. Ex. DDDD, at 467. People who undergo this therapy may test falsely positive for antibodies that are in the donor plasma. Id.

Respondent filed as reference 4 to Dr. Lancaster's third supplemental expert report (Ex. FFFF) a report of two cases by Abbas Bagheri et al., Psychogenic Unilateral Pseudoptosis, 31 OPHTHAL PLAST RECONSTR SURG e55 (2015), as Exhibit KKKK. The first case was a 21-year-old man with unilateral ptosis which occurred suddenly on his left side two weeks earlier. Ex. KKKK, at e55. He was treated by a psychiatrist and his symptoms spontaneously disappeared. Id. at e56. The second case was a 10-year-old girl with left upper eyelid ptosis for six months. She had stress in school. The doctors administered placebo intravenous saline injection and her symptoms almost completely disappeared, only to recur a few hours later. She was referred to a pediatric psychologist who treated her for conversion disorder. Two months later, she had complete recovery. Id. The authors note that conversion symptoms are clearly associated with psychological problems and environmental stresses. On clinical examination, the doctors could not find anything neurologically wrong. Id.

Respondent filed as reference 5 to Dr. Lancaster's third expert report (Ex. FFFF) a report of three cases by Jeanette W. Hop et al., Psychogenic Pseudoptosis, 244 J NEUROL 623 (1997), as Exhibit LLLL. The first case involved a 39-year-old engineer who developed ptosis of his left eye eight years before admission, without any other symptoms. Ex. LLLL, at 623. Episodes of severe drooping alternated with months without complaint. When administered a placebo of saline, his ptosis cleared. He had been under stress and underwent relaxation therapy. After three months, his ptosis completely disappeared.

The second case was a 30-year-old nurse who complained of eight months of tiredness, difficulty with walking, a funny sensation in her head (resulting three times in a brief loss of consciousness without any witnesses), swallowing difficulties, and concentration problems. For three months, she noticed a persistent lowering of her left upper eyelid which first started after a headache. When tired, she had diplopia while reading. All symptoms worsened in the evening. Id. Extensive medical and neurological investigation did not show any abnormality. Id. at 624. She was diagnosed with generalized somatization disorder and hysterical ptosis. The doctors recommended a program in behavior. But, two years and three hospitals later, her complaints and ptosis remained unchanged. After a pseudo-epileptic fit, she was not able to return to work. Id.

The third case was a 48-year-old woman who complained of episodes of abrupt involuntary closure of her left eye. At first, this resolved spontaneously after a few days, but during the next four months, the ptosis increased and she had double vision when looking to the left. She also complained of right calf pain, drowsiness, difficulty walking, transient attacks of loss of sensation in her left arm and leg, and episodes of transient loss of vision in both eyes, preceded by flashing lights. Testing did not show abnormalities. Her symptoms were unchanged until a follow-up visit three months later. Her husband had recurrent and bilateral facial palsies. The doctors opined the patient had some role copying contributing to her conversion disorder. She was treated for thrombocytosis which had been detected earlier in the year and six months later, her platelet count was normal. Her ptosis also almost completely disappeared without specific therapy. Id. The authors conclude that psychological factors contribute to conversion disorder. Id.

Petitioner filed as reference 7 to Dr. Steinman's seventh supplemental expert report (Ex. 198) a case report by Andrea Maier et al., GAD Antibodies as Key Link Between Chronic Intestinal Pseudoobstruction, Autonomic Neuropathy, and Limb Stiffness in a Nondiabetic Patient. A CARE-Compliant Case Report and Review of the Literature, 94 MEDICINE 1 (2015), as Exhibit 203. At age 28, the patient had her first symptoms of achalasia[152] and early satiety requiring her to eat more than five meals a day. Ex. 203, at 1. She had constipation with intervals of three or more days, leading to her using laxatives. She fainted several times and avoided standing upright for more than 15 minutes. Severe gastroesophageal dysmotility and dysphagia led to a weight loss of 15kg[153] in one and one-half years. Id. When she was 36 years old, intestinal dysmotility had advanced to chronic intestinal pseudoobstruction ("CIP") with complete paresis of the intestinal passage and severe abdominal pain. She had a percutaneous enteral tube and stoma inserted. Due to abnormal urinary retention, she had to catheterize herself four to six times a day. Dizziness and palpitations reduced her orthostatic tolerance to less than 10 minutes. Id.

[152] Achalasia is "failure of the smooth muscle fibers of the gastrointestinal tract to relax at a point of junction of one part with another; usually used to denote esophageal achalasia." Dorland's at 14.
[153] Fifteen kilograms is 33 pounds. CALCULATEME, https://www.calculateme.com/weight/kilograms-to-pounds/15 (last visited Mar. 22, 2019).

She developed dry eyes, dry mouth, dry, irritable skin with recurrent eczema, and difficulties in visual adaptation to darkness. Id. at 1-2. Occasionally she felt paresthesia and pain in her legs. Id. at 2. At the age of 37, she presented to the autonomic clinic of the authors with emaciation and spasms in her right leg. Her cranial nerves were intact except for an anisocoria[154] of one mm right < left eye and a reduced dilation of the right pupil in the dark. Her sensation was normal, including pain, light touch, vibration and proprioception. Deep tendon reflexes mainly of the right leg were increased. Id. Moving her legs passively was painful and difficult because her leg muscle tone was increased. Id. at 2-3. Testing of her autonomic nervous functions showed she had a right-sided Horner syndrome[155] of postganglionic[156] origin. Id. at 3. The patient had bilateral sicca syndrome.[157] She had detrusor hypocontractility with urinary retention and an enormous delay in the barium enema passage. Galvanic skin responses were delayed in hands and feet bilaterally. Head-up tilting at 70 degrees revealed a postural tachycardia with a heart rate increasing by 35 bpm. Her time upright was limited to five and one-half minutes due to presyncopal complaints. Id.

Nerve conduction studies, including somatosensory and motor evoked potentials, were normal. Repeated neuromuscular stimulation did not provide a basis for diagnosing the patient with myasthenia gravis or Lambert-Eaton myasthenic syndrome. However, the patient was diagnosed with stiff limb syndrome because of spontaneous grouped neuromyotonic discharges of motor units in her quadriceps and biceps femoris muscles of the right leg. Repeated measurements of her GAD65 antibodies were between 24 and 197IE/mL when the normal result should be <10IE/mL. The doctors administered IVIG at intervals of four weeks. Id. After three cycles of IVIG, her ability to stand rose from five to ten minutes and her weight stabilized. Id. at 3-4. Self-catheterization was reduced to two times a day. Id. at 4. This improvement remained for about three weeks, but then bladder retention recurred, her weight dropped again, and her gait unsteadiness and leg muscle stiffness increased. By adding prednisolone to the monthly IVIG, the patient improved.

The authors view this patient's condition as autoimmune. Id. They say GAD antibodies are considered organ specific and are usually associated with diabetes mellitus, although they can be found in nondiabetic patients such as the subject of the case report. They note that GAD antibodies are rare findings in patients with predominant enteric dysmotility. Id. (Petitioner did not have CIP, anhidrosis, spontaneous grouped neuromyotonic discharges, a brainstem lesion leading to Horner's syndrome, or urinary retention.)

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[154] Anisocoria is "inequality in diameter of the pupils." Dorland's at 93.

[155] Horner syndrome is "sinking in of the eyeball, ptosis of the upper eyelid, slight elevation of the lower lid, constriction of the pupil, narrowing of the palpebral fissure, and anhidrosis and flushing of the affected side of the face; caused by a brainstem lesion on the ipsilateral side that interrupts sympathetic nerve fibers." Dorland's at 1833.

[156] Postganglionic means "situated posterior or distal to a ganglion; said especially of autonomic nerve fibers so located." Dorland's at 1502.

[157] Sicca syndrome is "keratoconjunctivitis and xerostomia without connective tissue disease." Dorland's at 1848. Keratoconjunctivitis is "inflammation of the cornea and conjunctiva." Id. at 980. Conjunctiva is "the delicate membrane that lines the eyelids and covers the exposed surface of the sclera." Id. at 405. Xerostomia is "dryness of the mouth from salivary gland dysfunction." Id. at 2087.

Petitioner filed as reference 1 to Dr. Steinman's ninth supplemental report (Ex. 208), an article by Midhat S. Farooqui et al., Therapeutic Plasma Exchange and Immunosuppressive Therapy in a Patient with Anti-GAD Antibody-Related Epilepsy: Quantification of the Antibody Response, 30 J APHERESIS 8 (205), as Exhibit 209. One of the co-authors of this article is respondent's expert Dr. Lancaster. The women whom the authors describe was 23 years old, coming to the authors' hospital because of increasing seizure frequency over the prior few weeks. Ex. 209, at 9. Her seizures were generalized tonic-clonic typically preceded by an aura of anxiety and sometimes déjà vu. They usually occurred at night and lasted for about one to two minutes, followed by five minutes of confusion. Id. In the month prior to her going to the hospital, the patient had about one seizure a week which progressed to one per day. Id. at 9-10. The patient had been diagnosed with epilepsy four years earlier. Id. at 10. EEG showed frequent left temporal epileptiform discharges with intermittent left temporal slowing. A later MRI showed T2 hyperintensity in her left mesial temporal lobe and a small area of T2 hyperintensity in her right temporal lobe. Id. She had elevated anti-GAD antibodies in her serum, measuring 115,900 IU/ml at her highest level and, after completing five sessions of treatment with therapeutic plasma exchange ("TPE") and immunosuppressive therapy (mycophenolate mofetil for two days followed by oral prednisone), her lowest level of anti-GAD antibodies was 3,970IU/ml. Id. at 11. The normal level of anti-GAD antibodies is ≤5IU/ml. Id. at 10.

Over these five treatments with TPE, the patient was seizure-free. Id. The patient's decrease in GAD autoantibody burden and decline in serum reactivity to GAD antigens correlated with clinical recovery. Id. at 11. She went from one seizure per day to being seizure-free over the next month. After that month, however, her seizures recurred. Id. The authors conclude that the patient's seizure disorder had an immune-mediated component, noting that anti-GAD antibodies have been linked to a variety of neurological syndromes including stiff person syndrome, cerebellar ataxia, limbic encephalitis, and epilepsy. Id. at 11, 12. They also note that through immunostaining, they characterized the patient's GAD as the likely dominant neuronal autoantigen for this patient who had a progressive loss of GAD reactivity in her sera to neurons while she underwent TPE treatments. Id. at 13. The authors cannot conclude however whether TPE could be used as maintenance treatment as part of her long-term therapy. Id.

The undersigned fails to see the relevancy of this article to petitioner herein. Petitioner never had seizures. All her EEGs and brain MRIs were normal. She does not have any of the neurological syndromes with which anti-GAD antibodies are associated, i.e., stiff person syndrome, cerebellar ataxia, limbic encephalitis, and epilepsy. Moreover, her elevation above the ≤5IU/ml normal level of anti-GAD antibodies, ranging from 6IU/ml to 37IU/ml, are infinitesimally smaller that the patient's anti-GAD antibodies in this article, ranging from a high of 115,900IU/ml to 3,970IU/ml. Thus, petitioner has neither the neurologic illnesses with which anti-GAD antibodies are associated nor the high titers of anti-GAD antibodies which people with these neurologic illnesses have.

Respondent filed as reference 2 to Dr. Whitton's fourth supplemental expert report (Ex. MMMM) an article by Beth B. Murinson, Stiff-Person Syndrome, 10 THE NEUROLOGIST 131 (2004), as Exhibit OOOO. The author lists the characteristics of stiff person syndrome. Ex.

OOOO, at 132. The legs and spine are almost always affected. The patient will hold the leg in stiff extension making ambulation awkward. The most important clinical sign is increased lumbar lordosis. The abnormal postures are due to sustained muscle contraction. Abdominal muscles are frequently involved. Arms, when involved, may assume a flexed posture. Superimposed spasms can be overwhelming, making it impossible for a doctor performing a physical examination on the patient to bend or move the stiffened limb. The syndrome is associated with stress and results in numerous falls. These falls are described as statue-like or log-like. Id. Onset of stiff person syndrome is typically in middle age. Id. at 133. The classic EMG finding of stiff person disease is continuous motor unit activity. Id. at 135.

Petitioner did not have any of the characteristics of stiff person syndrome as depicted in this article. Petitioner complained of weakness. She would drop an arm in seconds. She would weave when walking, as if she were about to fall, but she would not fall. No doctor noted petitioner had lumbar lordosis. Her EMG was normal.

Respondent filed as reference 2 to Dr. Lancaster's third supplemental expert report (Ex. QQQQ) an article by Wei Hao et al., Epitope-Specific Glutamic Acid Decarboxylase-65 Autoantibodies in Intravenous Immunoglobulin Preparations, 9 TRANSFUS MED 307 (1999), as Exhibit SSSS. The authors report there are autoantibodies to GAD65 present in IVIG. Ex. SSSS, at 307. They tested six preparations of IVIG for the presence of GAD65 antibodies and found GAD65 antibodies in all six. Id. They also tested to see if the antibodies to GAD65 in IVIG were reactive in humans (as well as in rats and mice) and found that they were. Id. The authors note that the presence of GAD65 autoantibodies is not surprising since about one to two percent of healthy normal individuals may have GAD65 autoantibodies in their plasma. Id. at 309. They note that since IVIG is used as a treatment for patients with stiff person syndrome, a possible harmful effect may be caused by the administration of more GAD65 autoantibodies. Another possibility is a beneficial effect in generating an idiotypic immune response. They suggest screening commercial preparations of IVIG for GAD65 autoantibodies before treating patients with stiff person syndrome to detect the presence of GAD65 autoantibodies. Id.

Respondent filed as reference 3 to Dr. Lancaster's third supplemental expert report (Ex. QQQQ) an article by Jeffrey L. Kishiyama et al., A Multicenter, Randomized, Double-Blind, Placebo-Controlled Trial of High-Dose Intravenous Immunoglobulin for Oral Corticosteroid-Dependent Asthma, 91 CLIN IMMUNOL 126 (1999), as Exhibit TTTT. The authors compared the efficacy of high-dose IVIG treatment of severe, steroid-dependent asthma with placebo consisting of albumin and discovered no significant difference in treatment effects of IVIG and placebo. Ex. TTTT, at 126, 131. The authors did however note the severe adverse effects of IVIG treatment which can include aseptic meningitis and severe headaches. Id. at 131. Because of the cost of adverse effects of IVIG therapy, "clinical efficacy would need to be dramatic to justify such therapy." Id. They conclude, "In summary, in this controlled study, high doses of IVIG did not demonstrate a clinically or statistically significant advantage over placebo (albumin) infusions for the treatment of corticosteroid-dependent asthma." Id. at 132.

Other Filings

Respondent filed as Exhibit W the results of an 8K race held on October 17, 2009, which is almost eight weeks after petitioner received flu vaccine. Petitioner came in 179th out of 220 runners with a time of 56 minutes and 13 seconds. Ex. W, at 4. Eight kilometers is almost five miles, which means petitioner ran at a pace of about a mile in about 11 minutes.

Respondent filed as Exhibit X a page from a podcast edition of The Robert Scott Bell[158] Show, on the website Vactruth.com, dated November 5, 2009, which is 10 and ½ weeks after petitioner received flu vaccine. Mr. Bell interviews Dr. Rashid Buttar who states petitioner was now cured. Ex. X, at 1. After stating Dr. Buttar reversed a supposedly incurable neurological adverse event reaction, Mr. Bell refers the listeners to petitioner's own website which includes her name and "my story." Id. at 2.

Respondent filed as Exhibit Y pages from Age of Autism, NFL Cheerleader Disabled by 2009 Flu Shot on Road to Recovery, (Nov. 6, 2009), http://www.ageofautism.com/2009/11/nfl-cheerleader-disabled-by-2009-flu-shot-on-road-to-recovery.html. It shows petitioner in her Redskins uniform, smiling at the camera. It states:

> The vibrant, 25-year-old Washington Redskins Cheerleader Ambassador has a website to tell her story and keep well-wishers from around the world informed of her progress as well as to promote "true informed consent."

Ex. Y, at 2. The article says petitioner's story made headlines both in the US and abroad, with videos explaining her disorder attracting millions of viewers on YouTube. Id. It continues:

> The responses she has received have been overwhelmingly supportive, encouraging, and informative. Celebrity couple Jenny McCarthy and Jim Carrey helped point [petitioner] in the right direction through Generation Rescue, a non-profit organization dedicated to preventing and reversing autism.
>
> The treatments with Dr. Buttar at the Center for Advanced Medicine and Clinical Research in Charlotte, NC are working, and the results are nothing short of amazing. [Petitioner] can now walk and talk normally throughout the vast majority of the day and the seizures/convulsions have significantly decreased. Although her full recovery will take an undetermined amount of time, her family is now for the first time, convinced she will make a complete recovery. She is now more than ever driven by a desire to educate others to be informed of the potential side effects caused by vaccines and prevent others from suffering a similar fate.

[158] "Robert Scott Bell tackles the tough issues and shows no fear when confronting government and corporate bullies who would stand in the way of health freedom. You will be amazed by the amount of information about healing that is kept secret from you and what you can do to learn more about it." GCNLIVE, rss.gcnlive.com/RobertScottBell (last visited Feb. 25, 2019).

> Visitors to her new website [listing it] will find regular updates on her progress, helpful details on her treatment and valuable information on the importance of "informed consent" – truly knowing ALL of the options before making important medical decisions.
>
> "I set up the site to tell my story and warn people of the neurological side effects that can result from vaccinations," [petitioner] said, "Especially knowing that in the majority of cases, these stories are seldom heard outside of immediate families and friends." Visit [petitioner's website] for more information.

Id.

On October 21, 2011, petitioner left the following comment on a website called Operation Jack:

> I was injured too from a flu vaccine in 2009. They call it autism, but in reality it's really brain damage. I now exhibit a lot of the same cognitive issues that someone with autism would have. I have brain vasculitis and a shrunken cerebellum all from my mistake to become vaccinated. I however can still run and plan on running in this year's Operation for [sic] Jack race. . . .

Operation Jack, Picturing Regression (Aug. 24, 2011), http://www.operationjack.org/picturing-regression.

HEARING

For the four days of hearing, Robert J. Krakow, who was petitioner's first attorney, assisted Lisa A. Roquemore, who is petitioner's current attorney. Tr. at 2. For respondent, Debra A. Filteau Begley assisted Justine E. Walters. Id. at 3.

Petitioner's Testimony

Petitioner testified first. Id. at 18. She explained that she chose a French name for her last name at random in December 2012 because she was divorced twice and did not want to use her maiden name since, in 2009, the media paid a lot of unkind and unhelpful attention to her symptoms and a lot of people attacked her on social media. Id. at 19. She went public initially when a co-worker at AOL asked to do a story in the local newspaper he worked for and, from there, "it kind of took off." Id. at 20. Petitioner said her original reasoning for going public was she thought her batch of vaccine was tainted and she wanted to warn others that they should probably look into that particular batch. Id.

Petitioner went through her education. She was a junior studying for a bachelor's degree in finance and economics at night. She started working at AOL at 18 as a secretary. Id. She says she quickly moved up the ranks and became a communications marketing manager. She also worked at Morgan Stanley as a securities representative. Id. She holds a series 7, 66, 31

and an insurance license to trade annuities. Id. at 21. She also has a real estate license. She and her second husband divorced in February 2011. Id.

Petitioner described her health before receiving the flu vaccination in 2009 as excellent. She was training for a 5K race, which is about three miles, but she was not really able to do a lot of training given her work schedule. She was about to be promoted at work.

Petitioner said she had seasonal allergies in the spring and fall. Id. at 22. AOL had a vitality wellness program and she would submit health milestones, such as running 5Ks, in the hope of getting a vacation. Her seasonal allergies caused a sinus infection that became bronchitis. Id. She was still going to work 50 hours a week and going to the gym with her bronchitis and cough. Id. at 23. For a couple of months prior to the flu vaccination, the bulk of petitioner's activities were running three to five miles a week, exercising, and going to the gym. Id. at 25. In her spare time, she read or studied. Id. at 26. She eventually wanted to get back into the finance sector and start her own brokerage someday. Id. at 26.

Petitioner joined the Washington Redskins Cheerleader Ambassadors in April 2009. Cheerleaders were a dancing squad specifically meant to entertain on the field. However, cheerleader ambassadors were specifically meant to go around to the suite owners and interact with them. Id. To become an ambassador, you had to have a good background, good education, and speak very well because you were going to speak with CEOs of corporations and high-level executives in these suites. Id. at 26-27. You had to be able to articulate well and speak with them. Id. at 27. An ambassador needed a lot of intelligence, and the ability to do public relations and communication. Petitioner said she wanted to be an ambassador because she hoped to start a brokerage someday. She thought being an ambassador would be a good networking opportunity to find investors to eventually help her start her own brokerage. Her job as an ambassador was on Sundays, while she still worked at AOL. Id.

Petitioner said she got a flu vaccination in August 2009 and remembers the day because she and her then-husband were going to a barbecue that day and stopped at a Safeway to buy dessert. Id. at 28. She also felt bad that she was missing her younger brother's birthday that day. In addition, the flu vaccination would give her 200 more points for the AOL health wellness program. Id. She remembers her resting heart rate at the time was in the 60s. Id. at 29. Her blood pressure was usually around 100. Id.

Starting on September 3, 2009, she woke up and felt unwell, hot, sweaty, and nauseated. Id. at 29-30. Her symptoms worsened during the day. Id. at 30. She remembers it was September 3, 2009 because that was her and her then-husband's third wedding anniversary and they were supposed to go out to dinner. She did go to the dinner and powered through it. She went to work that day and powered through it. Id. After dinner, she was nauseated and felt really lightheaded. Id. at 31. Four people lived in the house with her: her husband, her brother, her sister, and a roommate. None of them was ill and no one at work was ill. Id. She ate dinner as she normally would, but she was too dizzy to drive home. Id. at 31-32. That evening, her nausea worsened and she was sweating. Id. at 32. She started feeling muscle pains as if someone were tearing a knife through all her muscles. Id.

On September 12, 2009 she went to the hospital. That morning, she ate a burrito for breakfast and water and an energy drink. After 20-30 minutes, she was sitting on the couch and felt nauseated, lightheaded, and really cold. She "kind of fainted sitting on the couch." Id. She got up and she got worse with more lightheadedness and dizziness. Id. She collapsed and her then-husband and roommate rushed her to urgent care. Id. at 33. This was a Saturday. She started shaking and had a hard time breathing. Id. She stated she had never fainted before her flu vaccination. Id. at 34.

At the hospital, she was started on IV saline and felt slightly better. She was diagnosed with rhabdomyolysis. After the IV, she felt much better. She had no problems keeping food down. She was on IV fluids for three or four days. Id.

The day after she was discharged from the hospital, she went back to work. Id. at 35. She felt pretty good on the day of discharge. A day later, her symptoms started to return. She felt extremely lethargic and fatigued and she did not have any appetite or very little appetite. A day or two later, she started having dizziness and almost fainted after she had eaten some grapes. Id.

On the second or third day back to work, she was sitting at her desk eating grapes and a coworker came up to her. Id. at 36. Petitioner was dizzy and lightheaded. The coworker grabbed her then-husband because he and petitioner worked in the same department together at AOL. Petitioner stood up and became lightheaded and dizzy. She had to sit down. Her then-husband took her to the hospital. Id. Between the two hospitalizations of September 12 and 17, she had no appetite at all and just ate very small meals. She had problems with nausea and almost vomited some of the time. Id. at 37. On September 17, 2009, she noticed heart rate issues. Her heart rate was a lot higher than it had ever been. Id.

In the hospital on September 17th, she was told she had a high ANA. Id. at 38. When she was discharged, her symptoms worsened. Id. at 39. It was harder for her to eat. Her appetite was almost completely gone. She had more dizziness and fainting. A few days after discharge, she had walking problems and speech problems along with fainting, nausea, and eating problems. Her walking problems consisted of feeling as if her legs were weak and buckling when she tried to get up. It started when she went to have a spinal tap. She was all right in the beginning of the morning, but halfway through the morning, her legs started to buckle and she was losing muscle control. Id. Her speech issues happened a day or two after that and she thinks it was stuttering. Id.

On September 26th, she went back to the hospital because of the walking issues and the dizziness. Id. at 40. A family friend who was a doctor said she could possibly have GBS and told her to go to the emergency room of a hospital to have them assess her. At the hospital, she started having breathing issues and they admitted her. Id. A nurse mentioned to an intern that petitioner had an orthostatic issue when she got up. Id. at 40-41.

Some of the hospital notes indicate petitioner was applying her makeup in the hospital. Id. at 41. Petitioner explained she had more strength in the morning and could do her showering, apply makeup, get ready, and had perhaps 30 minutes to an hour before she would start to lose

muscle control. She said she came into the hospital but was unable to walk and had a commode next to her hospital bed because she could not get up. She thinks what the hospital personnel were "probably insinuating" is that she had makeup on that she could not wash off because she could not get up to go to the bathroom. Id. She said she never at any time at any of her hospital visits had makeup with her. Id. at 42. She said she would have makeup on before she went to each hospital. Id.

Petitioner said that, in general, she typically had about 30 minutes to an hour in the mornings before any muscle issue "would kick in." Id. Gradually, that became maybe 20 minutes and, later on, she would waken and almost have complete muscle problems in just a few minutes. She had more strength in the morning. Id. Petitioner said that the personnel in the hospital visit of September 26, 2009 did not seem to know what was happening and told her they were stumped. Id. Her infectious disease doctor ran a series of tests and said there was no infectious disorder. Id. at 42-43. He said she should see a neurologist. Id. at 43.

Petitioner thought initially she had Lyme disease, but she tested negative for that. Her doctor prescribed an antibiotic anyway, which she took, but it did not help her symptoms. Id. She saw a psychologist during her hospital visit of September 26th to rule out conversion disorder as a diagnosis. Id. The psychologist said she did not have conversion disorder and "passed the test, so to speak." Id.

Petitioner explained the reference to her teenage bulimia in the psychologist's notes. Id. at 44. She only tried it because of friends but did not continue with it. Id. Petitioner had pain in her esophagus like heartburn, but a sharper stabbing pain that began in mid-September around September 16 or 17, 2009. Id. at 45-46. It occurred mostly after she ate. Id. at 46.

After her September 26, 2009 hospitalization, the doctors said they could not figure out what she had and she needed to follow up with her general practitioner. He said he was not really sure what was going on but she still had a high ANA. Id. He recommended she go to Johns Hopkins hospital for a diagnosis, which she did on October 2, 2009. At the time, she was still having a lot of eating issues. When she arose in the morning, she had maybe 10-20 minutes before she would have walking problems. They started to become almost a shaking movement. Id. The weakness combined with jerky movements. Id. at 47. After only 20-30 minutes after she ate, she would start getting dizzy and lightheaded. Then her speech became stuttering. She could not recall when her speech became more slurred. She also had a high heart rate. Id. She had one-word responses. It was hard to get out the words. She also had trouble breathing to make each answer. Id.

She also had trouble keeping food down. Id. at 47-48. When she tried to eat and move her tongue, it would produce other muscle symptoms, a kind of weakness and jerky movement in her neck. Id. at 48. When she attempted standing or sitting up, she would be dizzy and lightheaded from a meal. Id.

Petitioner said the personnel at Johns Hopkins told her she "was just having some kind of reaction to the vaccine and that, hopefully, it would get better within six weeks." Id. She saw a physical therapist at Johns Hopkins who saw her walking and said that type of dystonia is easily

treated by physical therapy and medication. Id. at 48-49. Petitioner said she was "kind of blown away" because no one had diagnosed her until the physical therapist said she had dystonia. Id. at 49. Petitioner testified that after petitioner told the Johns Hopkins neurologist what the physical therapist told her, he said petitioner should follow up with a neurologist and he would give her medication, Klonopin, to help with dystonia. Its generic name is clonazepam. The reason the Johns Hopkins neurologists gave for prescribing this was they thought she had some kind of reaction to the vaccine and the drug would help. But the main reason was that earlier, in the evening, they had prescribed Ativan to help her sleep. Id. When a phlebotomist came in to draw blood, she was able to speak very fluently after the Ativan without any problem. Id. at 49-50. In the morning, still on Ativan, she was able to walk briefly for a few minutes before she returned to having trouble speaking and walking. Id.

Petitioner explained that Klonopin is in the same family of drugs as Ativan, but an alternative because Ativan made her too sleepy to function during the day. No one else had a thought about an alternate diagnosis to dystonia. They just thought she had some reaction to the vaccination. They did propose she could have some kind of stress and should follow up with a general practitioner, a neurologist, a psychologist, and a physical therapist. Id.

The physical therapist told petitioner about sensory tricks. Tr. at 51. When petitioner got home, she went on the web and searched dystonia, finding a Mayo Clinic list of sensory tricks to try. For example, for speech, she placed her hand on her chin to alleviate the muscle spasm. Another sensory trick was to place pressure on her leg to alleviate some of the muscle spasms there. These sensory tricks worked initially for her. Running would alleviate the dystonia. Id.

Petitioner lost a considerable amount of weight. Id. Petitioner said that when the Johns Hopkins neurologist Dr. Anjail Sharrief saw petitioner's walking, Dr. Sharrief's jaw dropped and she looked like she had never seen anything like that before, but the physical therapist was just the opposite and knew what petitioner had as soon as she saw it. Id. at 52. Petitioner and her then-husband decided not to follow up with Dr. Sharrief when petitioner left Johns Hopkins and wanted to find someone with a little more experience. Id. Petitioner said the Johns Hopkins personnel told her Ativan would alleviate the symptoms of dystonia. Id. at 52-53. She had a lot of insomnia at the time. Id. at 53.

Petitioner said the publicity about her "took off into some kind of media circus." After the initial newspaper article with her former colleague, local news stations and national stations started calling her and she committed doing a few of those and "it just became its own beast." Id.

Petitioner went to see Dr. Cintron, a neurologist, in Reston, Virginia around October 15, 2009. Id. She said Dr. Cintron seemed to think she was having a reaction to the flu vaccination and it was causing a movement disorder. Id. at 54. Later on, he said it looked like it was also causing autonomic issues as well. Dr. Cintron did 30 minutes of a physical examination, watching her walk, doing different tests, having her whisper and sing (she was able to do both without having speech issues). Id. at 54-55. He recommended petitioner have IVIG treatment as well as a medication and she never did either. Id. Because of the media attention, Stan Kurtz

from Generation Rescue, "some whacky anti-vaccine group," approached her and offered to help cure her. In exchange, she would help them do a documentary. She said Stan Kurtz seemed convincing and believed he could cure her of all her symptoms. She felt it might be a better course of action since she had gone to so many hospitals and still did not have a treatment or an answer to what was going on. Id. She stated she did not think she had autism. Id. at 55-56.

On October 17, 2009, petitioner was getting ready to run a 5K or an 8K race. Id. at 57. She was going to run three to five miles. Her symptoms were probably at their worst at that point. She had trouble keeping food and water down. Her speech was horrible. She could get out only one word at a time and if she tried too hard, her speech became slurred. She was having walking issues. Whenever she ate or drank, she got lightheaded or dizzy. She did not want to do that race on that particular day. Id. It was raining and cold. Id. at 58. But she said she had committed to Generation Rescue and their documentary and they brought in film crews. Plus, she had committed to friends who were going to help her run the race. She did not want to back down on the commitment she made to them. Before this race, the last time she ran a race was probably in August when she did a three-mile run. Id. Petitioner states she did not run for exercise during that time period in September. Id. at 59. She discovered she could walk backward by accident. Id.

Petitioner states at this time, she had trouble chewing and swallowing even just liquids or moving her tongue around. Id. at 60. It would worsen her muscle problems. It was not so much the swallowing itself. It was the movement of her tongue. Id. Every time she tried to move her tongue around to swallow, she would have speech issues and breathing problems. Id. at 60-61. The undersigned asked petitioner if she were breathing through her mouth and she responded she would just stop breathing. Id. at 61. She was not choking. It was almost like the muscles with breathing stopped working and she could not physically get them to go on her own for a period of a few seconds. Her breathing "would just start working after a few seconds." Id. She would stop breathing when she was eating or doing something strenuous like overexerting her muscles, trying to talk when she could not talk or walk further than she could walk. This would bring on these brief periods when she stopped breathing. Id. The undersigned asked petitioner if she would faint and she said she could not quite recall if she would faint during these periods. Id. at 62.

Petitioner then consulted her supplemental declaration, Exhibit 139, page 9, paragraph 30, video #409_0237_01, because Dr. Lancaster commented on that October 17, 2009 video in his first expert report (Ex. H). Id. at 63. Petitioner said that her voice sounded more slurred than what Dr. Lancaster described. Petitioner described it as if the muscles in her tongue were not moving but were numb or more likely paralyzed. Id. at 67. She stated in paragraph 35 of her supplemental declaration that her throat felt paralyzed with swallowing. Id. at 68. As soon as she swallowed a liquid, she started feeling as if her lungs were paralyzed and she could not get them to move. Id. When petitioner's attorney asked her if it were her lungs, petitioner said, "Well, the throat." Id. at 68-69. She stopped breathing for a few seconds. Id. at 69. On October 17, 2009, it would be 10, 20 seconds. Id.

Ms. Roquemore asked petitioner if she agreed with Dr. Lancaster's comments on video #409_251_01 in her supplemental declaration, Ex. 139, page 12, paragraph 42. She said no. When she reviewed the video, she was having difficulty eating the strawberries which had to be cut up. She believes she had three or four bites before having trouble chewing and finishing the food. Therefore, she was not chewing easily and not swallowing easily. For a brief moment, she stopped breathing. Id. Then she started having shaking tremors. Id. at 70. Then her speech became slurred. She concludes there was a lot more going on in this video than Dr. Lancaster described in his expert report. Before all this, she would eat a whole pack of strawberries and not cut them up. Id.

Ms. Roquemore asked petitioner if she agreed with Dr. Lancaster's comments on video #409_258_01 in her supplemental declaration, Ex. 139, page 14, paragraph 49. Petitioner said this video shows her explaining how she was having shortness of breath when she walked or ate. Id. She would have stabbing pain in her ribs, hips, and knees. Id. at 71. In the video, she describes how she had trouble eating and she was losing a lot of weight because as soon as food "hit" her stomach, she felt sick. She also described in the video how when using her tongue to eat or swallow, that caused her symptoms to worsen and how in the hospital, when she was given IV fluids, she would feel a lot better and a lot of the symptoms would improve. She discussed how she had a high heart rate. Id.

Ms. Roquemore asked petitioner if she agreed with Dr. Lancaster's comments about a video she refers to in supplemental declaration, Ex. 139, page 14, paragraph 151 (without identifying the video number). The video shows petitioner eating a large salad and Dr. Lancaster said she did it easily. Petitioner protested that it was the opposite. She was trying to eat really quickly because she figured if she could eat fast enough, then she would get the food down before she had symptoms. Id. She said she could only get in three bites before she had trouble chewing. Id. at 72. Petitioner stated the video shows her becoming weak, and having shaky movements, tremors, and trouble breathing. When she tries to take another bite, she has weakness and is shaky. Her then-husband tries to help feed her and, several minutes later, she drinks water easily since she figured out that if she did not move her tongue and she was not chewing, she could get liquids down okay. Then she explains that her face feels paralyzed or the muscles feel as if they are not working. Later, others used a food processor to blend her salad so she could try to eat it later. Id.

Ms. Roquemore asked petitioner if she agreed with Dr. Lancaster's comments on video #409_0285_01 in her supplemental declaration, Ex. 139, page 16, paragraphs 58 and 59. Id. at 73. She said the video showed her nauseated and vomiting a small amount of pink liquid into a bowl. She said she had really weak leg muscles in that video. She denied spitting any food up. She said it was vomit. She was having a very hard time keeping food down. Id.

Ms. Roquemore asked if October 19, 2009 was the first time she saw Dr. Buttar and petitioner said yes. Id. at 73-74. She directed petitioner to look at her supplemental declaration, Ex. 139, page 17, paragraphs 64 and 65, and describe video #409_0326_01. Id. at 74. Petitioner said she was lying on some kind of examination table and personnel had fed her some baby food. She had not eaten a solid meal in days and was starving. Shortly afterward, she started feeling

dizzy, nauseated, and sick. She started vomiting and did vomit before this video began. Stan Kurtz ran out of the room to get Dr. Buttar. Id. The biggest issue she reported to Dr. Buttar was eating. Id. at 75. She also had weakness, especially with walking, periods of a few seconds when she would stop breathing, and speech issues. Id.

Petitioner thought that doing the race on October 17, 2009 worsened her condition quite a bit. Id. at 76. Some of the treatments Dr. Buttar gave her were hours and hours of some kind of IV solution whose contents she does not know. Occasionally, Dr. Buttar put her in the hyperbaric chamber for a period of time. When she received the IVs, she got stronger. She could eat without a lot of issues. She could eat a chicken sandwich. Id. She would go into the hyperbaric chamber after IV hydration and eat, drink, and speak fairly well. Id. at 77. Petitioner believes her symptoms were better because of the IV hydration. When she borrowed a hyperbaric chamber for about a year or two years after Dr. Buttar's treatments and got into the chamber without previous hydrations, she did not experience anywhere near the same effect as she did when she was on IVs at Dr. Buttar's or even at the hospital. Id. She was on IV solutions for eight hours a day when she was at Dr. Buttar's. Id. at 78.

When she left Dr. Buttar's or he decided to cut back her time on the IVs, she got worse. Her walking issues and difficulty eating came back. The video on October 21, 2009 showing petitioner eating a chicken sandwich was when she was still hooked up to an IV. Id. She was able to eat while on the IV. Id. at 78-79. She was able to walk as well. Id. at 79. This seemed to be true with hospital IVs, which is why she did not improve at Fairfax Hospital, because they never put her on any hydration or IVs at all. She had walking problems the entire time she was there. Id. At this point, the undersigned asked petitioner's counsel why petitioner needed immunoglobulin intravenously if just fluid with either saline or glucose was enough to make petitioner better, and asked her to have Dr. Steinman respond to that question. Id. at 80-81.

After petitioner left Dr. Buttar's and he stopped the IVs, she went back to the hotel and thought that she could eat the same way she had earlier in the day, but found out quickly that she could not. Id. at 82. All her stomach issues came back. Stan Kurtz call Dr. Buttar to see her than night. Id. Initially, Stan called to have Dr. Buttar bring more IVs, but Dr. Buttar could not. Instead he brought some drops to see if he could treat her issue after eating. Id. at 82-83. Initially, Dr. Buttar put on 10 or 15 drops on her arm because her arms were really weak. Id. at 83. Several minutes later, petitioner noticed her speech slightly improved but then it went back to being slurred. Dr. Buttar applied more drops again to try to get her speech back. In the video, petitioner described a burning pain because of the drops but it felt as if the burning was her muscles tightening and then releasing. At the same time, her speech would come back and then become kind of slurred. "Back and forth. Whatever the drops were doing was not holding." Id.

Petitioner said she had a falling out with Dr. Buttar during a 20/20 interview in December. Id. at 84. She did not want to do it. She was not feeling well, but Dr. Buttar reminded her that he had treated her for free. She said she was forced into doing the interview and Dr. Buttar wanted her to stick "with this ridiculous script about talking about autistic kids," and she refused to do that. When 20/20 asked her if she were cured, she told them no, not at all, and she also said things that they did not put in the final cut. She testified one of the things that

did not go in the final cut was her statement that she was getting worse. That is when Dr. Buttar stormed out of the interview room and stopped all her treatments. Id.

Petitioner returned to Dr. Cintron around October 20, 2009 because she was not getting better with Dr. Buttar's treatments and his treatments were starting to get a little bizarre. Id. at 85. She thought it was time to cut ties with Dr. Buttar and find an actual good doctor. Dr. Cintron prescribed Valium to help her walk. He also referred her to Dr. Tanenbaum, a cardiologist, because she was having trouble with high heart rate and blood pressure. Id. Petitioner said that Dr. Cintron believed she had a vaccine reaction causing movement disorder issues and autonomic issues. Id. at 86. She took a stress test and, when she got close to her maximum heart rate, she felt really dizzy, lightheaded, and overall not well. She got weak and collapsed from the lightheadedness. The doctor told her she had some kind of neurocardiogenic syncope. Id.

In late October, early November, her voice settled on a speech impediment that sounded like a foreign accent. Id. at 86-87. It was better in the morning but, by the end of the day, it was more pronounced. Id. at 87. However, the slurred speech would return when she ate too much. In February 2010, she had a transcranial Doppler where the technician pushed a wand against both her carotid arteries. Petitioner was lying down and not feeling well from the procedure. When the technician finished, petitioner tried to sit up and became so dizzy and lightheaded that she started almost falling over and felt as if she were going to vomit. The technician called in Dr. Cintron and he said petitioner had some autonomic issues and he wanted her to go to the hospital. Id. She saw Dr. Nayak on February 12, 2010 after the stress test with Dr. Tanenbaum because Dr. Nayak was a specialist in autonomic instability. Id. at 88. Dr. Nayak did an EKG and checked her heart rate and blood pressure. He touched her carotid arteries and she started getting lightheaded and dizzy again. Id. Dr. Nayak believed the cause of her problems was the vaccination and what happened in the beginning might have been some sort of GBS-type of issue and she should have been on IVIG or plasmapheresis early on. Id. at 88-89. Dr. Nayak said she should see an electrophysiologist. Id. at 89.

Petitioner saw Dr. Christine Cosgrave, a psychologist, who told petitioner she had a neurological issue and should see a neurologist. Id. Petitioner saw Dr. Atiga, whom Dr. Nayak had recommended, on March 23, 2010. Id. at 89-90. Petitioner had to go running in order to be able to keep any food down. Id. at 90. She still had dizziness, lightheadedness, and a high heart rate. Dr. Atiga put her on Norpace to increase her exercise tolerance. Her problem was when she stopped running, she got so dizzy and lightheaded that it was dangerous to stop exercising. Id. He also prescribed Midodrine to get her blood pressure higher. Id. at 90-91. Dr. Atiga told her he believed the vaccination caused her problems. Id. at 91. She started taking Neurontin in December 2010. She started having trouble with Valium because it made it difficult for her to breathe at times and made her a little bit weaker. After a couple of days of taking Neurontin, she did not have any more issues with walking. Id. An increase in the dose in March improved her walking greatly. Id. at 91-92. She did not have the stuttering in her speech as much on Neurontin. Id. at 92. Her foreign accent did not improve on Neurontin, but her dizziness and

lightheadedness slightly improved. Neurontin helped slightly with her voice issues. It helped mostly with her walking and jerky movement of her neck.

Inside Edition did an interview outside Walmart as an update in mid-January 2010. Id. It was early in the morning and she was heading from a fabric store to her car when the interviewer approached her in the parking lot. Tr. at 93. Her walking was good. She had a little bit of a walking issue from a slightly torn quadriceps. She had the speech impediment in the video. She explained she walked sideways in the video to get to her car because it was parked close to another car. Id. She went to an emergency room the day after the video. Id. at 94. The hospital put her on a couple of IV hydration saline to help with her fainting and getting lightheaded which had occurred during the transcranial Doppler when the technician pushed on her arteries. Id.

Petitioner said she noticed in April or May 2010 that she had issues with walking in extreme heat, which to her would be 85 degrees. Id. at 96. Her walking would become weak, she would have trouble speaking (slurring), she would get lightheaded and dizzy, and she would start having trouble breathing. If she exercised, usually running, she became able to eat more food or a normal moderate diet without having nausea, vomiting, or lightheadedness, but it was exhausting to keep doing that. Id. Dr. Atiga told her to keep her meals small and space them throughout the day. Id. at 97. She found she could eat more calories if she exercised. She moved to California to get out of the heat in Washington, DC. Id.

Dr. Atiga did the first tilt-table test on petitioner on July 15, 2010. Id. at 98. Petitioner said a few minutes into the test, she started having trouble speaking and her speech became slurred. Then the technician gave her a spray in her mouth and, quickly afterward, her speech became even worse and she had a brief period of trouble breathing. She shook and got dizzy. That is when the technician completed the test. Petitioner said she had slurred speech for close to an hour afterwards. The technician would not let her go until her speech improved. Id.

Dr. Atiga thought petitioner might have a problem with blood flow in her brain and she looked up top doctors specializing in autonomic issues, making an appointment with Dr. Yan-Go at UCLA. Id. at 98-99. Petitioner said Dr. Yan-Go thought she was having some autonomic issues and did her own assessment, recommending petitioner see a gastroenterologist about her eating problems and have an esophageal test for motility for swallowing issues. The motility test showed that her swallowing was normal. Id.

Petitioner saw Dr. Ghassemi, a gastroenterologist, at UCLA in August 2010. Id. at 100. Petitioner testified that Dr. Ghassemi thought her issues were autonomic and within the neurologic specialty. He recommended Sandostatin, whose generic name is octreotide, an injection one self-administers before meals. Id. She obtained the medication and it worked perfectly. Id. at 101.

She found an autonomic lab in Ohio to get a second tilt-table test as Dr. Yan-Go had recommended. Id. at 102. She had a lot of the same issues with the second tilt-table test as at the first one, but not as severe. She noticed she had a high heart rate, her hands and feet were freezing, and she had a little slurred speech. Id. Dr. Atiga called her and told her she had POTS, grade two. Id. at 103. She had testing to get documentation that she needed Sandostatin which

consisted of blood pressure measurements at intervals while she ate. Petitioner believes there was an 18-point drop in blood pressure. Id. Petitioner said that Dr. Wilkinson, her cardiologist in Seattle, believed her blood pressure, heart rate, dizziness, trouble eating, speech impediment, i.e., the whole condition, was due to the vaccination. Id. at 105.

On July 29, 2011, petitioner saw Dr. Grubb at the University of Toledo, who is supposed to be the top specialist in autonomic issues and POTS. She waited almost a year to see him. Id. Dr. Grubb had a frank discussion with her that none of her symptoms was treatable by medication and that her life would not return to the way it was before. Id. at 106. He told her he was not surprised by her divorce because a lot of spouses leave after someone becomes ill. He also said petitioner would probably never be able to have children because pregnancy would be too difficult and dangerous, and she would not have the physical capability to take care of children if she were able to have them. She thinks they spoke for almost two hours. Id. Petitioner said that Dr. Grubb told her the vaccine caused this and he had seen similar cases in the past. Id. at 107. Dr. Grubb told her that the vaccine caused some kind of autoimmune response that attacked her nerves controlling her autonomic system. He drew pictures for her. He prescribed Mestinon to help the autonomic issues and a vitamin to see if it would help her cognitive issues. Id. Dr. Grubb told her that Mestinon was used for myasthenia gravis, but off label, for autonomic issues. Id. at 108.

Petitioner said she tried Mestinon for a day or two and did not notice that it affected her autonomic issues although her speech was a little better. But since it was expensive and not improving her autonomic issues, she stopped taking it. She could not afford to spend money on a medication that did not improve her symptoms dramatically as she had hoped. Id.

Petitioner's general practitioner ordered a SPECT scan to see if it would identify the cause of her issues with dizziness and cognition. Id. at 108-09. The result was that she had issues with blood flow in her brain and doctors needed to rule out lupus and a second autoimmune disease. Id. Her gastroenterologist Dr. Elmer Chang recommended petitioner continue Sandostatin but, after a year or 18 months, the Sandostatin stopped working. Then Dr. Chang recommended a feeding tube. Eventually her feeding issues became impossible to manage. Id. She was vomiting and could not keep food down. Id. at 110. In 2013, she had a feeding tube inserted. Earlier, a gastric emptying study showed severe gastroparesis which Dr. Chang said meant food was not moving out of her stomach as fast as it should and that is why she was vomiting. Id.

In 2012, petitioner had another ANA panel and the result was higher than her first ANA panel, the first being 1:40 and the second being 1:320. Id. at 114-15. In January 2012, she saw Dr. Lyden, a neurologist, at Cedars-Sinai. Id. at 115. He said petitioner had nystagmus. Id. Dr. Leyden said her reflexes were not present. Id. at 116. He also said her pupil size was irregular. Id. Petitioner provided him with all her medical records. When she saw Dr. Lyden a couple of days later, he had completely changed in his tone toward her. Id. He no longer wanted to see her and dismissed all of his earlier findings. Id. at 118. Dr. Lyden said petitioner needed to see a psychologist. His demeanor completely changed. Petitioner stated Dr. Lyden must have seen

something in the medical records about the media. He had not seen her medical records before her first visit to him, but only after that first visit and before the second visit. Id.

Then petitioner saw Dr. Olek, a neurologist specializing in MS. Id. at 119. Petitioner said she had been stable for two and one-half years, but starting in January 2012, she was much worse. Her fingers turned completely white anytime she was near cold. She could not eat very well, despite being on $5,000 a month medication. She had a dropped foot. She saw Dr. Olek in February 2012. He thought she might have autoimmune dysautonomia or autoimmune autonomic issues. He thought she might have central nervous system lupus. Id. She was in extreme pain from her stomach extending out so far she looked pregnant. Id.

Petitioner went to see Dr. Gee, a neurologist. Id. at 120. He was thinking she might have autonomic issues and wanted to test for lupus. Id. at 121. He told her the vaccine caused her issues. Id. She saw the rheumatologist Dr. Wallace in spring 2012. Id. Petitioner testified that Dr. Wallace thought she had an autoimmune autonomic disease and that she might have myasthenia gravis. Id. at 122. Petitioner stated that when she worsened in 2012, she thought she would try Mestinon again that Dr. Grubb had prescribed and found it improved all of her muscle issues. She no longer had problems with walking, strength, breathing, or speech. Id.

In November 2013, petitioner went to the hospital emergency department because she was very weak and had a cold plus a fight between her cat and her dogs produced stress. She had stridor. Id. Her perception of stridor is that her vocal chords were collapsing and not allowing air to come in. Id. at 123. She noticed every time she became ill, her foreign accent came back and she would have breathing issues at night and walking issues. Her muscle weakness became worse. The hospital ED noticed her heart rate was extremely high and that she had a heart arrhythmia called a long QT. Id.

Dr. Hung, a cardiologist, told her that a long QT is extremely dangerous and can be quickly fatal. Id. at 124. The doctors suspected autoimmune autonomic disease was perhaps exacerbating arrhythmia in her heart. Id. Dr. Gee told her his sister died of lupus in her twenties and that the progression of petitioner's disease was very severe and affecting a lot of critical systems. Id. at 124-25. He said petitioner should fight as long as she could and see if IVIG would extend her life. Id. at 125. Petitioner testified that Dr. Gee tested for myasthenia gravis, but she was either on steroids or IVIG and he gave up trying to get lab results. Dr. Gee said petitioner had myasthenia gravis because she responded to Mestinon and to IVIG. Id.

Petitioner went on IVIG every four weeks after this hospital visit and it improved her muscle weakness and stomach slightly. Id. at 127-28. But her breathing and muscle weakness were not fully under control and she still had dizziness, fainting, and problems with heat and breathing. Id. at 128. Because her symptoms on IVIG were not fully resolved, she saw Dr. Sheean in San Diego. Id. Dr. Sheean said the vaccination caused petitioner's rhabdomyolysis that led to her myasthenia which led to her autoimmune autonomic neuropathy. Id. at 130. Dr. Sheean said, in the alternative, the vaccine triggered all three at the same time. He thought petitioner's IVIG dosage was too low and should be raised "to mop up the antibodies" her body was making every month. Id. He also said her contraceptive pills were making her diseases

worse and she needed to avoid getting pregnant. Id. at 130-31. This is what led petitioner to have a hysterectomy. Id. at 131. Dr. Sheean wanted petitioner to take a low-dose of a chemotherapy drug. He said all the IVIG was doing every month was "just mopping up the mess of the antibodies" and he wanted her to take the chemotherapy drug "to kind of stop the immune system completely." Id. She did not want to go on the chemotherapy drug until she figured out whether to donate her eggs to her cousin or what options she had. Id.

Petitioner is still on IVIG. Id. at 132. Her muscles weakness is improved. She had occasional breathing issues in the middle of the night while sleeping. However, the new dosage of IVIG is so high that she has more problems eating and relies more heavily on the feeding tube to get through the several hours between breakfast and dinner. Id. She still has dizziness on the new dosage. Id. Dr. Gee said that high-dosage IVIG worsens autonomic problems. Id. at 133. Petitioner does not know if she still has rhabdomyolysis. Id. Petitioner stated she does not see a doctor separately for autonomic dysautonomia. Id. at 135. Dr. Sheean treats her myasthenia gravis and Dr. Gee treats her myasthenia gravis and autonomic dysautonomia. The IVIG would be helping her dysautonomia if it were given at a lower dose. Id.

Dr. Gee said petitioner would be on this treatment for the rest of her life unless she tried the chemotherapy. Id. at 136. Even though she had a hysterectomy, she still has her ovaries and she has not decided what to do with her eggs. Id.

On cross-examination, petitioner admitted that she created a calendar of September to November 2009 a week before the hearing. Id. at 140. She put it together from memory. Id. Her testimony about her conversations with her doctors that was not contained in any of her medical records is all based on petitioner's memory. Id. at 141. Respondent's counsel had the calendar marked as Exhibit SSS, and a seven-page document entitled "Medical Treatment: Doctor and Personal Statements and Frustrations" marked as Exhibit TTT. Id. at 142. Petitioner testified she thought her then-husband created Exhibit TTT. Id. at 143. Petitioner found it a couple of weeks before the hearing. Id. It is written from her then-husband's perspective. She thought her then-brother-in-law might have put some of Exhibit TTT together as well. Id. Respondent moved into evidence a series of reflections on respondent's expert Dr. Lancaster's opinion about the videos as Exhibit VVV. Id. at 152.

Petitioner thinks she filed 40 hours of videos, half with Dr. Buttar and half taken before she saw Dr. Buttar. Id. at 165-66. She first talked to Stan Kurtz in mid-October 2009. Id. at 166. It was soon after NBC and Fox News aired on October 15, 2009. Some of the videos showed petitioner before she arrived at Dr. Buttar's office. Id. Stan Kurtz arrived at her home the day before the 8K race petitioner did on October 17, 2009. Id. at 166-67. When Stan Kurtz arrived, he came with a doctor and a video crew. Id. at 167. Stan Kurtz said in exchange for the video footage, he would pay for her treatments with Dr. Buttar who he said was making progress in treating vaccine injuries. Id. At some point, Stan Kurtz put petitioner in contact with Jenny McCarthy and petitioner did a video chat with Jenny McCarthy. Id. at 168. Stan Kurtz videotaped petitioner at the beginning and end of the 8K race. Id.

Petitioner said her cognitive issues began with recollection, mathematics, and reading. Id. But most of those problems improved greatly since she has been on IVIG. Id. at 169. She said her cognitive issues made it harder for her to access information and relay it to people in a timely fashion. She believes it was Dr. Cintron who diagnosed her with dystonia. Id. She told him that the physical therapist mentioned dystonia. Id. at 170.

Petitioner denied a number of details in a history she stated her then-husband gave to Inova Fairfax and Loudon hospitals and (with her then-brother-in-law) to Dr. Buttar, including: fainting, chest pain, uncontrollable laughing, trembling, hot flashes, drinking a glass of wine and two mixed drinks over a four-hour period, constant sweating, sore throat, the correct date of the vaccination, greenish-yellow mucus, taking a decongestant and two Aleves, body aches most severely at spots injured while working out (hip, muscle, bicep pull), blacking out. Id. at 171-81. Petitioner said she had trouble speaking at the time and her then-husband did all the talking to the doctors. Id. at 181.

Petitioner denied the accuracy of Dr. Mannon's notes on September 27, 2009 when he wrote petitioner's speech was quite clear. Id. at 188. Petitioner insisted she had speech and stuttering issues. Id. She said the psychologist recognized she had to whisper because she had trouble talking. Id. Petitioner stated Dr. Mannon got a history from her then-husband because of her problem talking either because she was stuttering or slurring. Id. at 189. She said, "In my opinion, there was no clear speech. It was not clear." Id. Petitioner denied she had had a flu-like illness. Id. at 190. She said she had had symptoms she never had before. Id.

Petitioner said that her then-husband filled out the VAERS report even though her name is on the completion line. Id. at 191. When petitioner heard that the VAERS report identified "parent" as the identity of the relationship to the patient, she thought her former father-in-law might have filled it out. Id. at 192. Or maybe both her then-husband and his father filled out the VAERS report. She believes that because the wrong date of vaccination is on the form. Id. Petitioner denied that she had written she had a sore throat, nasal congestion followed by fever, body aches, chills, and a headache. Id. at 193. She denied having a sore throat, nasal congestion, a headache, or a fever. She guesses her former father-in-law filled out the VAERS form. Id. Petitioner denied even knowing what VAERS was. Id. at 194.

Petitioner denied writing in the VAERS form that she had flu-like symptoms, fainting, violent convulsions, walking as if she had MS, and entire body shaking. Id. She said her former father-in-law was an English major and he does not write anything in the third person if it is supposed to be in the first person, which is why the language in the form sounds as if she had written it, i.e., "my," and "I". Petitioner said the symptoms in the VAERS form are not consistent with what her actual symptoms were at the time (the form is dated October 7, 2009) and given her former father-in-law's "anal retentiveness to English being proper, I believe that he filled this out and put it into the first person, especially since a lot of these symptoms are not what I remember and reported, the vaccine date is wrong, and he has parent as relationship to the patient in here." Id.

Petitioner said she did not recall having a sore throat or congestion. Id. at 196. She does remember allergies. She said that all the histories given to doctors and hospitals that, after the flu vaccination, she had a cold, a cough, a sore throat, a fever, and mucus are wrong. Her then-husband gave those histories because she had trouble breathing and speaking and she relied on him to be the communicator. She did not have the capacity to speak up and correct him. Tr. at 195-96.

Respondent's counsel asked petitioner if she were too ill on October 7, 2009, the date of the VAERS form, she was also too ill to do anything on the computer. Id. at 197. Petitioner replied that she could work minimally on the computer, but she had trouble eye tracking and trouble reading. She said she was more focused on trying to eat and alleviate her symptoms which was why her then-father-in-law and then-brother-in-law were there, to help with administrative things. She could check e-mail but not transcribe a whole report. She could look at things in the morning. Id. Petitioner said she discovered a way to read by using a piece of paper to track a computer page. Id. at 198. She would put the piece of paper on the screen. But this was so exhausting and time-consuming that she could not read all the e-mails she used to read at work. She was just able to do enough to see if her mother e-mailed her, but not enough to fill out an entire VAERS form and that is why her former father-in-law and former brother-in-law were in town. Id. She does not remember if she used the piece of paper on the computer in early or late October or early November. Id. at 199.

Regarding putting on her makeup, petitioner said she would have a short period of time in the morning when she did not have any symptoms. Id. And when the period of 30 minutes ended, she would have difficulty walking. She told doctors she had uncontrollable tremors. Id. Originally, she had an hour in the morning before her symptoms would "kick in." Id. at 200. Then that time period shortened to about 30 minutes, then 20 minutes, and so forth. In the morning, during that period, she would do what she did as if she were going to work: take a shower, get ready, and put on makeup. Then she would eat breakfast. That is when she started having tremors and walking problems. After that period, she would have speaking issues after she ate. Id. Petitioner said her tremors were limited to her neck and hands. She does not recall if her entire body had tremors. Id.

In the morning, she was able to lift her hands to her face to apply her makeup, including mascara and foundation. Id. at 201. She washed her hair every three to four days. Id. She also applied eyeliner, but she had to sit down and use her elbow resting on the table to apply the mascara, eyeliner, and eyeshadow. Id. at 202. Petitioner had a vanity and put her elbow against the vanity to apply her makeup. Id. She did not need to hold the opposite eye taut while she applied the eyeliner and mascara because she "was really good at it." Id. at 203. She used liquid eyeliner and found a trick for applying mascara where if she just held the brush and blinked into it, she would apply the mascara. Her eyebrows are so thick, she did not need eyebrow pencil. Id.

Petitioner said that she usually sat down in the shower even before she became ill. Id. at 204. It was just easier for her. She would lean her elbows against her knees and turn her head down to reach the shampoo and minimize the muscle strength to wash her hair. She still uses

those techniques. Id. She would air dry her hair. Id. at 205. She had a hair dryer attached to the wall like a hand dryer in a gym, but this was an ionizer. The only thing she found difficult was brushing her hair when it was wet. Id. That was to get all the knots out. Id. at 206. She put her elbow on the vanity and tried to use both hands to get out the knots or would ask her then-husband to help. She still uses these techniques today. Id.

Petitioner said her then-husband did not want her to drive. But once she got stronger with the medication Dr. Cintron prescribed, she would go out when her then-husband was at work. As seen in Inside Edition, she went shopping at a fabric store and her then-husband did not know she was out. She told the interviewer of Inside Edition not to let her then-husband see the interview. She had "ways of dealing with the weakness, just clever." Id.

Petitioner said that September 12, 2009 was when she had a lot of weakness because it took longer than usual for her to get ready to go out to breakfast with her then-husband. Id. at 207. She clarified that when she said she sat in the shower, it was not on a chair but on the floor of the shower and she would use the handles of the stall shower to lower herself and raise herself. Id. Petitioner said September 23, 2009 was when she started having walking issues. Id. at 208. The handles in the shower were an inch or two inches above her head when she sat on the floor of the shower. Id. at 209. The shampoo was located on the floor of the shower and petitioner used body wash instead of soap. The body wash was on the floor of the shower, too. Id.

Petitioner testified her symptoms were at their worst when she went to Dr. Buttar's clinic in October 2009. Id. at 210. She rapidly deteriorated after her 8K race on October 10, 2009. When she arrived at Dr. Buttar's clinic, she was too weak to walk. After she ate, she would have tremors. Id. Petitioner had tremors associated with eating or drinking, usually in the mid-morning and afterward. Id. at 210-11. She described her tremors as a shaking of her neck and sometimes her arms. Id. at 211. "It was kind of like a weakness that wasn't – like a weakness in the muscles that was making it tremor is kind of how I felt." Id. At the time, she and her then-husband referred to them as seizures even though they did not know what they were. But in retrospect, they were definitely more like tremors and "not seizures." Id. Petitioner thinks her then-husband was reporting them as seizures and saying they happened 60 times a day because petitioner was not able to count how often they happened. Id. at 211-12. "We were counting something that we thought were seizures when they were really just tremors." Id. at 212.

When she reported seizures to doctors, she meant tremors. Id. Petitioner thinks the tremors in early or mid-September 2009 were not as significant as they were closer to her visiting Dr. Buttar's clinic. Id. at 213. The tremors worsened quite significantly after she ran the 8K race. Id. If she tried not to move and tried not to eat or drink, she did not have any tremors. Id. at 214. They were just little bursts of weakness. In early September, she ate only twice a day. Id. She did not have an appetite. Id. at 214-15. Later in September and in October, she was concentrating on keeping food down. Id. at 215. It did not matter what kind of food she ate. She would be more likely to have a longer period of tremors toward the end of the day than earlier and she would have more nausea and vomiting at that time of day. Id.

In October 2009, she had five or ten minutes after she ate before getting lightheaded and dizzy, and if she did not lie down, "the tremors would come on." Id. at 216. After that period, she would get tremors or weakness in her arms and neck if she put anything in her mouth and tried to chew or swallow. If she did not move, she was fine. But the moment she tried to eat more or use those muscles again, the tremors would return. Id. At some point, she had to eat all her meals lying down. Id. at 217. Some of the videos show her lying down and eating. But moving her tongue around to swallow what she chewed would cause tremors and issues with muscle weakness. She thinks being wheeled backward after looking at the carpet in Dr. Buttar's office started making her feel sick and the tremors "started to happen." Id.

Petitioner said while she was at Dr. Buttar's clinic, she was not allowed to speak. Id. at 218. All they allowed her to do was make gestures. They did not want her speaking because they thought speaking would worsen her symptoms. She agreed that the videos in Dr. Buttar's office in mid-October 2009 showed her having a lot of tremors. The tremors started when she tried to shake Dr. Buttar's hand or when they fed her baby food and she immediately got sick and lightheaded and started having tremors. Id. Any sort of muscle movement would cause tremors which she attributes to a weakness in her arms and neck. Id. at 219. Petitioner thinks the weakness caused the tremors. Id. at 220. Drinking cold liquids would cause the tremors. Id.

Respondent's counsel asked petitioner if the Inside Edition news broadcast that aired on October 16, 2009, Ex. GGG, was a good example of her forward-walking gait problem. Id. at 221. Petitioner agreed that that is what her gait looked like before going to Dr. Buttar's office. Id. at 222. It was after she was discharged from Johns Hopkins Hospital on October 5, 2009 and before she saw Dr. Cintron October 15, 2009. Id. at 223-24. Her weakness began on September 23, 2009, but the jerky gait did not begin until closer to her going to Johns Hopkins. Id. at 224. When she went to Johns Hopkins, she had the same gait as the Inside Edition broadcast showed. Id. at 225. Her gait and speech problems completely resolved when she ran. She also found that she could walk normally sideways. Id. She recalled that one of the sensory tricks that worked was touching her hand to a pressure point on her leg and, if she tied a belt to her leg, she was able to walk forward mostly normally. Id. at 226.

On October 2, 2009, petitioner had a lot of difficulty talking without stuttering when she went to Johns Hopkins. Id. at 227. Although the notes at the hospital said she could be understood quite well if she were singing, petitioner does not remember that. Id. She said only Dr. Cintron asked her to sing for him and that was well after she went to Johns Hopkins. Id. She does recall that she could speak well while whispering at Johns Hopkins. Id. at 227-28. She learned that whispering enabled her to talk normally at Inova Fairfax Hospital September 26, 2009. Id. at 228. She said she did not correct the wrong information her then-husband gave to the doctors at Inova Fairfax because they did not come to see her after she was admitted as a patient and she was limited to the nurses. Id. at 229. It was still cumbersome to try to whisper to someone an entire history especially the doctor who interviewed her. Id. at 229-30. Whispering relieved her stuttering. Id. at 232.

Petitioner arrived at Dr. Buttar's office on October 19, 2009. Id. at 234. Her symptoms were at their worst. Id. at 235. She does not know why Dr. Buttar did not take her to the

hospital. Dr. Buttar immediately hooked petitioner to an IV and put her feet in a foot bath that he said would help chelate her. Id. On October 20, 2009, the day after she arrived at Dr. Buttar's clinic, petitioner had a remarkable improvement in her symptoms. Id. at 236. Following the IVs, she got much better. She was able to communicate with an entirely normal voice. After the rounds of IVs and maybe the chelation, she was able to speak and walk pretty well. She told Dr. Buttar she felt the best she had felt in the prior month. She told him she was starving and requested chicken. Dr. Buttar told her that her improvement would likely be transient and she would need more treatment. Id. On October 20, 2009, she was able to walk out of Dr. Buttar's office and return to her hotel room. Id. at 236-37. She could walk and talk normally. Id. at 237.

When she returned to her hotel room and ate, she had a recurrence of her symptoms. Id. Stan Kurtz called Dr. Buttar. Id. at 238. She was lying down on the couch and had slurred speech and muscle weakness in her arms. Id. Dr. Buttar came and put liquid TD ("transdermal") DMPS on her arms. Id. at 238-39. Ten minutes later, after Dr. Buttar repeated the drops on her arms, her speech returned and then went back to slurring. Id. at 239. Briefly, her speaking improved. She said she had a burning sensation moving throughout her body. Id. Her muscles were spasming, tightening, and constricting, going from her lungs to her neck or throat and face and then it dissipated. Id. at 239-40. After the burning, she had difficulty speaking again. Id. at 240. Dr. Buttar put more drops on petitioner and the speech eventually completely resolved, but she never got up and does not know if the weakness, dizziness, and nausea improved. Id.

Eventually, petitioner did get up after the food had moved out of her stomach in an hour or an hour and one-half. Id. at 241. She used the drops one or twice afterward and believes they resolved some of the muscle weakness or the tremors. Id. at 242.

There is a video of petitioner in the hyperbaric oxygen therapy chamber with Stan Kurtz talking to her, saying this was the first time he had heard her voice. Id. at 243. Dr. Buttar's IVs allowed her to speak. Id. She does not know why Stan Kurtz said this was the first time he had heard her voice. Id. at 246. There were periods of time when her symptoms improved before she went in the hyperbaric oxygen therapy chamber. Id. at 248.

In mid-September 2009, she had cognitive issues where she could not do math or process conversations. Id. She noticed it more toward the end of her visit to Dr. Buttar's clinic when she no longer had to worry about walking or talking. Id. Her cognitive skills improved slightly in late December 2009 or early January 2010 when she had occupational therapy. Id. at 249. The most dramatic of the cognitive improvement came with IVIG. She thinks that when her speech issues resolved, she was left with a speech impediment which sounded like a foreign accent. Id. On November 6, 2009, her stuttering and inability to talk resolved, but she could not move her tongue properly and her speech sounded like an accent. Id. at 250.

On the second day of testimony, on redirect, petitioner said that Dr. Buttar would start her treatment on a few hours of IV before she ever went into the hyperbaric oxygen therapy chamber. Id. at 264. They did not give petitioner oxygen in the hyperbaric chamber. Tr. at 265. She was inside the chamber for an hour. Petitioner found it hard to say whether there was any

difference being in the chamber. She always felt better after Dr. Buttar gave her several hours of IVs. Id. He said she could eat and drink in the hyperbaric chamber. Id. She noticed that her mercury levels were barely out of the normal range and she does not know what mercury was coming out of her system. Id. at 267.

On recross, petitioner stated that she believed at the time that when Dr. Buttar gave her the IVs, she started to feel better. Id. at 268.

Dr. Steinman's Testimony

Dr. Lawrence Steinman testified next for petitioner. Id. at 271. He is board certified in neurology. Id. at 272. He stressed that he is not an advocate. Id. at 273. He said even though he appears only for petitioners in the Vaccine Program, he does not appear when a case does not have merit. Id. He is a specialist in multiple sclerosis and does animal experimentation. Id. at 278. At the time of the hearing, Dr. Steinman was treating patients no more than 25 percent of the time. Id. at 281. He does full-time practice for two to four weeks a year as he is then in charge of the neurology ward, but because of his other activities, he stopped doing outpatient work when he became chairman of the department of immunology. In his earlier years, he split his activities between clinical and research 50-50. At the time of the hearing, he was doing research 75 percent of the time. Id. He said everything he does involves teaching and learning. Id. at 282.

For the first 35 years of his career, the National Institutes of Health has been the main source of his funding at Stanford. Id. He was elected to the National Academy of Medicine and the National Academy of Sciences. Id. at 283. In 2011, he received a lifetime achievement award from the International Federation of MS Societies named after Martin Charcot, who discovered or first described MS. Id. at 284-85. Sigmund Freud was one of Charcot's students and the two of them are responsible for what then was called hysteria but is now termed conversion disorder. Id. at 285.

Dr. Steinman said he talks a lot about molecular mimicry in this case, but he does not have a known molecular mimic that he can identify:

> THE WITNESS: I talk a lot about molecular mimicry, and I don't have a known molecular mimic that I can identify. There might be one, but I haven't been able to identify it, including something in the influenza vaccine that Petitioner received . . . .

Id. at 286. He thinks there might be one, but he has not been able to identify it. Maybe it would be on an autonomic acetylcholine receptor in a ganglion. Dr. Steinman testified that the autonomic nervous system is myelinated and a reaction to myelin can cause dysautonomia. Id. He stated there is a spectrum in inflammatory neuropathies between GBS to inflammatory demyelinating neuropathy and dysautonomia. Id. at 287. He thinks a common mechanism can link the pathogenesis. He thinks that if the autonomic nervous system is myelinated and there is damage to autonomic myelin, that can also damage the underlying axon and underlying nerves to cause dysautonomia. Id.

The undersigned asked if dysautonomia can occur without damage to the myelin of the autonomic nervous system. Dr. Steinman said it probably can be an immune response against the ganglionic acetylcholine receptor. Id. Petitioner in this case was not tested for it. Id. at 288. If petitioner had been tested for an immune response against the ganglionic acetylcholine receptor, the antibody is positive only about 50 percent of the time. Id.

The undersigned asked if myasthenia gravis is a dysautonomic disease. Dr. Steinman replied myasthenia gravis is a disease against the acetylcholine receptor at the neuromuscular junction. Someone with many of the symptoms petitioner described can have myasthenia gravis. Dr. Steinman said there are a few other antigenic targets, e.g., muscle-specific kinase, but he does not know that any of these other antigenic targets is a direct molecular mimic of anything in influenza that cross-reacts with myelin. Id. He thinks that when someone has both dysautonomia and myasthenia gravis, a connecting mechanism probably exists. Id. at 289.

Dr. Steinman said he has done a lot of research on myasthenia gravis and published on it. Id. at 290. He stated myasthenia gravis is probably one of the best understood of all autoimmune conditions. Some interesting molecular mimics between other viruses, but not influenza virus, exist. He cited as an example herpes virus and acetylcholine receptor as a molecular mimic. Id.

Dr. Steinman said that, as a practicing neurologist over the years, he has seen people with dystonia. Id. It is a whole specialty unto itself. Id. at 291. He has seen a whole catalog of abnormal movements. He testified he has been involved in diagnosing dysautonomia. He is the general attending physician on the neurology practice and often gets patients with dysautonomia. The neurology practice sees dysautonomia occurring when a patient has the inflammatory neuropathy GBS. The neurology practice also sees dysautonomia associated with other neurologic diseases. Sometimes, the neurology practice sees people presenting primarily with dysautonomia. To Dr. Steinman, dysautonomia is one of the less well understood areas of neurology. Id.

Dr. Steinman said he has dealt with diagnosing people with POTS. In POTS, the patient has difficulty regulating postural changes, i.e., getting up from a supine position, and the patient's heart will race. Id. Dr. Steinman called the condition "fascinating." Id. at 292.

Dr. Steinman stated he has seen and treated many patients with myasthenia gravis. Nearly all his patients with myasthenia gravis have had altered speech with stridor and a voice that gets weaker during the day with nasal speaking. He has also seen patients with MS, especially those with impairment of their cerebellar pathways who have speech that is very hard to understand.

He refers patients with gastroparesis to a gastroenterologist although he has seen patients who have gastroparesis as a manifestation of dysautonomia. Id.

Dr. Steinman interprets someone speaking with a British or Australian accent as really having nasal speech. Id. He said one can hear foreign accent in people who have dysphonia from dystonia. Id. The undersigned asked if when people have a cold, they sound like Laurence

Olivier, to which Dr. Steinman responded that he was nodding his head in agreement and he does not have full answers, but it is intriguing. Id. at 294.

Dr. Steinman's opinion is that 10 days after receiving flu vaccine, petitioner had symptoms of rhabdomyolysis followed by a series of neurological symptoms that he would describe as ultimately dysautonomia. Id. at 295. His opinion is that flu vaccine was the trigger of both the rhabdomyolysis and the dysautonomia. He does not link the rhabdomyolysis to the dysautonomia, but he thinks flu vaccine triggered both. Id. When the undersigned asked Dr. Steinman if he thought petitioner had myasthenia gravis and that the flu vaccine caused it, Dr. Steinman responded that he thinks dysautonomia caused petitioner's major problem and very fine doctors think she has myasthenia gravis and he agrees with them. Id. at 295-96. He said petitioner has been on treatments for myasthenia gravis. Id. at 296.

Dr. Steinman testified that petitioner's case is one of the most complicated because of several different diagnoses which some superb treating doctors made. Id.

Dr. Steinman defined autoimmune inflammatory neuropathy as a condition in which the immune system attacks myelin, predominantly in the peripheral nerve or nerve roots. Id. at 297. Its most classic manifestation is GBS. One of the manifestations of autoimmune inflammatory neuropathy is dysautonomia. Dr. Steinman would put at one end of the spectrum inflammatory neuropathy dysautonomia and at the other end of the spectrum GBS without dysautonomia or with some dysautonomia. He views that spectrum as the same disease. Id. He stated in autoimmune dysautonomia, the manifestation of symptoms is related to the autonomic nervous system, including heat intolerance, cold extremities, excess sweating, feeling too warm, problems with gastric motility, problems with maintaining blood pressure when getting up from a chair or from a supine position. Id. at 297-98.

The undersigned asked Dr. Steinman if all dysautonomia is autoimmune. Id. at 298. He responded probably not. He said there are likely to be some inborn errors of the autonomic nervous system that can cause dysautonomia. Id.

Dr. Steinman then said the following:

> THE WITNESS: And I have to comment, after hearing the Petitioner yesterday, even after seeing her on videotapes, and certainly after reading the – all the medical records, there is nothing like face-to-face meeting with a human describing their [sic] problems.
>
> I spent yesterday, as we all did, hearing the Petitioner, and it's so very different from reading PDFs on a computer and watching videotapes on a little screen

Id. at 302, 303.

Dr. Steinman said petitioner's onset of symptoms was September 3, 2009 with weakness, dizziness, and cold-like symptoms. Id. at 304. On September 11, 2009, she had emesis. On

September 12, 2009, she had decreased appetite, nausea and vomiting. Id. She also had syncope, shaking because she felt cold, weakness, and talking in one-word answers. Id. at 305. Dr. Steinman allowed that having cold symptoms may be a symptom of an allergy or an infection. Id. at 305-06. If Dr. Steinman accepted that petitioner had a cough, sore throat, a runny nose, and fever, it could be an infection or an allergy. Id. at 306-07. If he were to consider that petitioner had both an infection and the flu vaccine, he would say the flu vaccine aggravated the viral infection. Id. at 308-09. He would say the flu vaccine was a substantial factor. Id. at 309.

The undersigned said to Dr. Steinman that in order for the vaccine to aggravate her cold, the cold symptoms would have had to have preceded the vaccination, but here the vaccination preceded the cold symptoms. Id. at 310. Dr. Steinman agreed that the temporal positioning in this case was hard for asserting aggravation because how could flu vaccine aggravate the cold symptoms if the vaccination occurred before the cold symptoms. He then said he felt a lot more comfortable with saying flu vaccine was a substantial factor. Id. He feels very strongly that petitioner does not have conversion disorder. Id. at 311.

Dr. Steinman said that having the flu affects autonomic behavior, from feeling cold, clammy, and sweaty at the same time as having decreased appetite and feeling sleepy while feeling faint and sometimes fainting. Id. at 312. Dr. Steinman said that petitioner's weakness on September 3, 2009 would certainly be a manifestation of myasthenia gravis. Id. at 313. Talking in one-word sentences could be a manifestation of myasthenia, which petitioner manifested on September 12, 2009. The cough could be a manifestation of respiratory weakness, also myasthenia gravis on September 12, 2009. Fatigue on September 12, 2009 could also manifest myasthenia gravis which involves feeling tired and weak. Id. On September 17, 2009, petitioner's shortness of breath could be due to muscle weakness from myasthenia gravis. Id. at 314. Dr. Steinman added profound bilateral lower extremity weakness followed by minimal weakness was due to myasthenia gravis. In addition, lower extremity weakness was due to myasthenia gravis. Id.

Dr. Steinman explained an elevated ANA is sometimes associated with systemic lupus erythematosus. Id. Sometimes, it accompanies myasthenia gravis. Id. at 314-15. He admitted petitioner does not have lupus. Id. at 315.

In Dr. Steinman's second supplemental expert report, Ex. 108, he states that autoimmune inflammatory neuropathy is also known as GBS and GBS is widely known to include autonomic nervous system symptoms, citing a Medscape article. Id. at 315-16. Dr. Steinman explained that the autonomic nervous system is myelinated. Id. at 316. He thinks petitioner had a demyelinating disease restricted to the autonomic nervous system which did not affect the myelin of her peripheral nerves. Id. at 317. Dr. Steinman believes dysautonomia is a variant of GBS even without CSF changes and without high levels of autoantibodies that bind to ganglionic AChR. Id. at 318.

Dr. Steinman refers to petitioner's cardiologist Dr. Walter Atiga and his relating the timing of petitioner's symptoms of vasovagal or neurocardiogenic mechanism to the date of flu

vaccination. Id. at 319. Dr. Atiga thought petitioner had an unusual form of neurocardiogenic syncope because when she did more physical exertion, her symptoms improved and she could actually eat, but when she did not exercise, she was worse. Id. at 320. Dr. Atiga's notes that petitioner had normal heart rate and blood pressure in response to an upright tilt table. But because petitioner's symptoms worsened with nitroglycerin, Dr. Atiga thought petitioner might have a form of cerebral syncope involving dysregulation of cerebral blood flow. Id. Dr. Steinman thinks the results of petitioner's tilt-table test was a positive indication that petitioner had dysautonomia. Id. at 321.

The undersigned asked Dr. Steinman why petitioner got better on IVs with just saline or glucose, but not immunoglobulin. Id. at 322. Dr. Steinman said he had "a very elegant explanation of why she's feeling better." Id. He said people with POTS are a little bit hypovolemic. Id. They do better with an increase in blood volume. Id. at 323.

While Dr. Steinman was discussing newly-admitted medical articles on the point that intravenous infusions help people with POTS, petitioner sank to the floor and started growling. At this point, the undersigned told the court reporter to go off the record. Id. at 329.

While we were off the record, the undersigned asked Ms. Roquemore if the undersigned's law clerk should call 911 and she said yes. The undersigned's law clerk called 911. All three doctors in the hearing room, including Dr. Steinman, kept their seats. The undersigned asked Dr. Steinman, since he was petitioner's expert, to go to her and see how she was doing. The EMTs arrived and took petitioner away on a gurney to Medstar Georgetown University Hospital. After that, Dr. Steinman retook his seat as the testifying expert. The undersigned went back on the record and the following colloquy occurred:

> THE COURT: Dr. Steinman, can you, because you were the one administering to her until the EMTs arrived, can you describe what was going on?
>
> THE WITNESS: We noticed that she had developed stridor, very noisy breathing. She was actually moving air reasonably well. She didn't look cyanotic from her nail beds, her pulse was fast, but regular, and she was having a lot of problems. The EMTs came and she wrote down on a piece of paper that normally she would take Mestinon, a drug for myasthenia, in this situation.
>
> I first asked does she need epinephrine, and because of the stridor, whether it was pharyngeal edema, and she said no, very definitively. And the EMTs are taking her now. They didn't have to put in an IV or intubate her immediately. So, they felt comfortable just moving her in a sitting position. …
>
> THE COURT: Is the attack that you saw that she had something that is a part of her illness? Is this myasthenia gravis, is this part of dysautonomia, or what is it?

THE WITNESS: Well, you can certainly see this in myasthenia gravis, stridor could be part of the dysautonomia, but my first two guesses were it was either her myasthenia, and she wrote Mestinon on a piece of paper that she would normally take but she didn't have it with her. The second thing that came to mind was she was having some kind of pharyngeal edema, laryngeal spasm for which she would want to take epinephrine, but throughout the whole thing, she moved air. It sounded and looked horrible, but her nail beds and her lips looked like she was moving air reasonably well.
…

THE COURT: Because conversion disorder has been raised as an issue, do you think what you just saw and helped her with, trying to cope with, was an actual episode of stridor, or was this something that she performed, and it wasn't actually a physical problem?

THE WITNESS: It would be pretty hard, in my opinion, to do stridor. It takes a huge amount of effort for that long as an act. It really did sound like bona fide stridor, and it was going on for a long time, but she was moving air. She didn't turn cyanotic. So, that's -- I don't think it was a manifestation of a conversion disorder, but I imagine all of these stresses here created a lot of anxiety, but it looked like – well, it didn't look like, it was stridor. If the person can do that as part of the conversion disorder, it's pretty impressive.

THE COURT: And you mentioned, and you've mentioned it numerous times, that she was "moving air." Is that unusual with stridor? Do you normally not move air with stridor?

THE WITNESS: Well, the biggest concern I had is that she would turn blue and then we would have to do something – it's even really hard to give mouth to mouth with somebody in stridor because you can't move them by their vocal cords. So, there's that crazy thing that we all hear about, and you would never want a neurologist to try it, of sticking a pen without the ink cartridge through their cricothyroid membrane. I mean, I wasn't going to do it, I would need a young guy to do that. But in all seriousness, she did seem to move air okay, in terms of not turning cyanotic.

THE COURT: No, I'm not suggesting that you gave her the improper treatment, I'm just, because of my own ignorance, asking, is it unusual to move air if you're actually having an attack of stridor?

> THE WITNESS: Most people who have stridor are able to move air okay. I mean, where I've seen it really get bad is little kids with croup, you know, they sound like they're barking, and they start getting cyanotic. She in the sitting up position, which she preferred, was moving air okay.
>
> THE COURT: Did she also throw up?
>
> THE WITNESS: Just dry heaves.

Tr. at 329-36.

Dr. Steinman resumed his testimony on direct, going through the medical notes of Dr. Cintron, petitioner's neurologist, and Dr. Grubb, her internist and cardiologist. Id. at 341-42. Dr. Grubb notes autoantibodies against peripheral acetylcholine receptors, but Dr. Steinman said there is no evidence that petitioner had antiganglionic acetylcholine receptor antibodies. Id. at 342-43. Dr. Steinman's theory is based on the fact that autonomic ganglia have myelinated fibers going into them and damage to the myelin could cause a transmission defect. Id. at 343. Dr. Steinman mentions Dr. Lyden and his initial view that petitioner might have severe dysautonomia. Id.

Dr. Steinman said these doctors were giving it their best shot with a lot of difficulty in putting everything together and that is what he was doing as well. Id. at 344. He said he would like to know if petitioner had antibodies to acetylcholine receptor before she took IVIG because, if so, he would not be able to use his theory of molecular mimic to myelin. Id. at 345. He cannot find a molecular mimic in flu vaccine to acetylcholine receptor. Id.

Dr. Steinman notes that Johns Hopkins did not diagnose petitioner with GBS. Id. at 347. Dr. Wallace, a rheumatologist, thought petitioner had Raynaud's of the brain. Id. at 347-48. The undersigned asked Dr. Steinman, "You can't have Raynaud's of the brain, can you?" to which he replied, "I don't think so." Id. at 348.

Dr. Steinman said he did not know who Dr. Sheean was before this case and looked him up. Id. at 349. Dr. Steinman thinks Dr. Sheean's four possible diagnoses are close to his own thinking. Id. at 351. Dr. Sheean believes that vaccines can cause myasthenia gravis. Id. at 356. Dr. Steinman states that the autonomic nervous system, sympathetic and parasympathetic, is myelinated in its preganglionic portion. Id. at 357. Thus, he says his arguments about how the immune system attacks myelin after exposure to flu virus leading to autoimmune dystonia are relevant. The autonomic ganglia received input from demyelinated preganglionic fibers and the location of this attack disrupted ganglionic transmission. Id. Dr. Steinman repeated that dysautonomia is a variant of GBS. Id. at 358.

Dr. Steinman said, regarding rhabdomyolysis, that there are case reports of both flu virus and flu vaccine causing it. Id. at 359. He said one could make the argument that demyelinated nerves can innervate skeletal muscle as collateral damage. Id. at 359-60. Dr. Steinman said he would not invoke the occurrence of rhabdomyolysis in this case in order for him to opine that flu vaccine caused petitioner's dysautonomia. Id. at 360. He said petitioner's case is complex and

he does not need the "step" of rhabdomyolysis. Petitioner had so many things going wrong, i.e., rhabdomyolysis, autonomic neuropathy, some doctors thinking she had myasthenia, not just the anti-acetylcholine receptor ganglionic response, but also the nicotinic acetylcholine response. Petitioner was getting hit at all levels. He asked why this was happening in one individual, why in addition she has ANA antibodies, which may be a predecessor of something like lupus, but we do not have a full explanation. Dr. Steinman said petitioner is "quite rare." Id.

The undersigned told Dr. Steinman that the undersigned did not regard GBS as a predicate to finding flu vaccine caused petitioner's dystonia, and the undersigned did not consider her to have GBS as no one diagnosed her with GBS. Id. at 361. The undersigned assumed Dr. Steinman was referring to GBS only as an analog. Id. at 362. The undersigned asked if the undersigned understood his testimony correctly that he was not keying in dysautonomia to GBS as if it were a Jack and Jill causation. He said, "That's right." Id. He said if there were any relationship, it is at the complete other end of the spectrum. If they were Jack and Jill, they are only connected by opposite ends of the spectrum. The undersigned asked if that spectrum were called autoimmunity and Dr. Steinman agreed he would call the spectrum "autoimmunity." Id.

As for petitioner's having seizures in the videos, even though petitioner denied in her testimony that she had seizures and said she confused seizures with tremors, Dr. Steinman said based on the videos, he still does not know if petitioner was having seizures. Id. at 365. However, he did think the videos showed petitioner had dystonic tremor. Id. at 367-68, 369. He thought it more unusual for petitioner to lose the tremor when she ran forward. Id. at 368. Dr. Steinman said he wants to see her in an examining room and put her through various paces and he cannot have that liberty watching videos. Id. at 371. He said that some of the videos having to do with publicity created a negative impression in him. Id. He did not like that happening and this is the part of the case that he is unhappy with. Id. at 371-72. He was not happy that it was on national news programs and is a medical mystery. Id. at 372. He was unhappy that petitioner was engaging voluntarily in national publicity. Id.

Dr. Steinman regards petitioner as having the same dystonia as in the Marsden article (Ex. 170), in which the subjects could not walk normally but could run, dance, or walk backward. Id. at 373. He said that stridor can be a manifestation of dystonia. Id. at 374. Returning to the Marsden article, Dr. Steinman mentioned that Dr. Marsden was the world's expert in neuromuscular disease in the second half of the 20th century, and dystonia is a really puzzling phenomenon. Id. at 375-76. Marsden's article was on torsion dystonia which is not the same as dysautonomia, but someone can get dystonia in dysautonomia. Id. at 376. Dystonia is a subsection of dysautonomia. Id. He said the old term for torsion dystonia was dystonia musculorum deformans because people thought something was wrong with the muscles. Id. at 376-77. Eventually, the muscles can become hypertrophied and atrophied because of awkward positions. Id. at 377.

Stanley Fahn's article (Ex. 129) describes a clinical variance of idiopathic torsion dystonia in 1989. The abnormal movements constituting dystonia are diverse with a wide range in speed, amplitude, rhythmicity, torsion, forcefulness, distribution in the body and relationship

to rest or voluntary activities. Id. Fahn mentions a child who has idiopathic dystonia in one leg, but only when walking forward; it could be absent when running or walking backward. Id. at 378. Dr. Steinman said this is what petitioner had. Id.

Dr. Steinman stated that many of petitioner's symptoms overlap with myasthenia gravis. Id. at 379. He noted that Fluzone, the flu vaccine petitioner received, has been associated with a case of myasthenia gravis. Myasthenia gravis is associated with dysautonomia. Id. Shukla (Exs. 102 and 103) assessed autonomic function in patients with myasthenia. Id. On orthostatic tests, myasthenia patients and the control group both had a rise in heart rate, and systolic and diastolic blood pressure. Id. at 379-80. However, the myasthenia patients had a significantly higher rate and duration of the rise than the control group did. Id. at 380. Similar differences were observed in the hand grip test. No difference was observed between the two groups on the parasympathetic test. The authors concluded that myasthenia gravis patients have sympathetic hyperreactivity which could cause some hemodynamic abnormalities. Id. Dr. Steinman said that dysautonomia and autoimmune dysautonomia can affect in some cases parasympathetic fibers. Id. The most famous parasympathetic innervation[159] is the vagus nerve.[160] Id. at 381.

Dr. Steinman testified that there is evidence of dysautonomia from the tilt-table tests and the QT interval abnormalities, i.e., cardiac arrhythmia. Id. at 382. In addition, the gastric motility study showed gastroparesis. Id. at 383. Petitioner did not have decremental repetitive stimulation as electrophysiologic evidence of myasthenia. She did not have acetylcholine receptor antibodies. Nevertheless, Dr. Sheean thought she could have myasthenia and he is a pretty compelling person because of his neuromuscular specialty. IVIG treatment could give a false positive on tests for myasthenia and could also suppress the immune response so that the tests were negative. Id. He said that IVIG is a popular drug because it is effective in a number of diseases ranging from GBS to other autoimmune conditions. Id. at 388. It is also approved to treat inflammatory neuropathy and chronic inflammatory neuropathy to a certain extent. Its side effects are headaches. Id.

The undersigned asked Dr. Steinman if petitioner improved because she was able to enter the courtroom walking forward and did not have any tremors because the IVIG treatment was working. Id. at 389. Dr. Steinman said it is possible. Id. at 390. The undersigned asked Dr. Steinman why he hesitated. He responded that he did not know enough about the whole contemporary picture to know whether to attribute petitioner's doing better to the IVIG. He thought petitioner gave a compelling history in the first day of hearing. Her voice was strong and she held up for a long time on the stand. He thought she was doing marvelously, but then

[159] Innervation is "1. the distribution or supply of nerves to a part. 2. the supply of nervous energy or of nerve stimulus sent to a part." Dorland's at 942.

[160] The nervus vagus is the "vagus nerve: tenth cranial nerve; *origin*, by numerous rootlets from lateral side of medulla oblongata in the groove between the olive and the inferior cerebellar peduncle; *branches*, superior and recurrent laryngeal nerves, meningeal, auricular, pharyngeal, cardiac, bronchial, gastric, hepatic, celiac, and renal rami, pharyngeal, pulmonary, and esophageal plexuses, and anterior and posterior trunks; *distribution*, descending through the jugular foramen, it presents as a superior and an inferior ganglion, and continues through the neck and thorax into the abdomen. It supplies sensory fibers to the ear, tongue, pharynx, and larynx, motor fibers to the pharynx, larynx, and esophagus, and parasympathetic and visceral afferent fibers to thoracic and abdominal viscera; …; *modality*, parasympathetic, visceral afferent, motor, general sensory." Dorland's at 1261.

she collapsed the morning of his testimony. He said, "I don't know how well she's doing or not. It seems like a big mess to me." Id.

Dr. Steinman does not think the technician who did the brain scan finding marked reduction in watershed profusion bilaterally could diagnose lupus or antiphospholipid antibody syndrome from it, but he said that was just his doubting personality. Id. at 391-92. He thinks the scan adds to the evidence that petitioner has something autoimmune going on. Id. at 392. Dr. Steinman said he is not so impressed with these kinds of brain scans, but it was conducted not at a highly respected institution that knows how to do these things. Id. at 393.

The long QT is part of the abnormality of the autonomic nervous system. Id. The long QT is a cardiac induction arrhythmia. Id. at 394. It sometimes has no consequences and sometimes can produce very serious medical consequences. The undersigned asked Dr. Steinman if he had any explanation for petitioner's testifying that when her doctor increased her dosage of IVIG, her myasthenia gravis got better but her dysautonomia got worse. Id. Dr. Steinman said no, he did not. Id. at 395.

Dr. Steinman posited that Fluzone containing H_1N_1 induces not only anti-myelin basic protein antibodies, but also antiganglioside antibodies. Id. at 403. Although Dr. Steinman opines there is a 15 to 20 percent binding level between a mimic in Fluzone to myelin, there is no mimic in flu vaccine to the acetylcholine receptor. Id. at 409. However, the preganglionic axon is likely myelinated and that is the connection to the 15 to 20 percent binding level between the mimic in Fluzone to myelin. Id. at 410. The best argument he can muster is that flu vaccine targets the myelinated preganglionic axon and induces an immune response with inflammation in the preganglionic axon, inducing damage in the ganglion itself. Id. This is his answer for how flu vaccine causes dysautonomia. Id. at 411. He cannot say what is a mimic between the components of flu vaccine and ganglionic acetylcholine receptor. Id. at 411-12.

Dr. Steinman gave an onset interval of 10 days for petitioner's dysautonomia symptoms after receiving flu vaccine on August 23, 2009. Id. at 415. He relates petitioner's weakness, dizziness, decreased appetite, nausea, and vomiting to dysautonomia. Id. at 416-17. These symptoms occurred on September 12, 2009. Id. at 417. That is more like an onset of 20 days. He attributes the same symptoms to myasthenia gravis as to dysautonomia and thus the onset of myasthenia gravis is the same 20 days. Petitioner also had rhabdomyolysis in that same period. Id. In attempting to link petitioner's later diagnosis of gastroparesis, Dr. Steinman said once an autoimmune autonomic nervous system disorder begins, it was "perfectly reasonable that other manifestations will occur." Id. at 419. Dr. Steinman agreed with Dr. Lancaster's point in Exhibit H that petitioner's results for her gastric emptying study done on March 30, 2012 could have been due to all the medications that she was taking affecting gastric motility. Id. at 424. But, Dr. Steinman said that it does not mean she did not have gastric emptying problems. He admitted petitioner is a very complex patient. Her gastric emptying study result was abnormal and she was on a lot of medicines. He said, "We're stuck." Id.

Dr. Steinman said petitioner has a variety of diagnoses. Id. at 427. He stated he said directly and indirectly that myasthenia gravis was not his favorite diagnosis but a significant

expert Dr. Sheean thinks petitioner has myasthenia or Lambert-Eaton Syndrome and Dr. Steinman puts a high weight on an expert treating physician. Id.

Dr. Steinman stated, "We're not saying that it's GBS here, we should make that clear." Id. at 430. He said we are not talking about GBS-related autonomic dysfunction. Id. at 434. We are talking about the other end of the spectrum. We do not think it is GBS. Id. He agrees with Dr. Lancaster that there is no evidence of petitioner having antibodies to myelin basic protein. Id. at 435. Most normal people have responses to myelin basic protein (to HFFK). Id. He said that autoimmunity in myelin basic protein has not been studied extensively in autoimmune dysautonomia. Id. at 436-37. When Ms. Roquemore asked, referring to Dr. Lancaster's comments in his expert report (Ex. H), what are Dr. Steinman's thoughts "that although GBS was initially suspected by a few physicians, her preserved reflexes, normal EMG, NCS studies and normal CSF proteins all rule out the diagnosis?" Dr. Steinman responded, "I don't think I base my opinion on GBS." Id. at 637.

Dr. Steinman does not think petitioner got better from chelation therapy, but from the fluid loading. Id. at 439. He attributes petitioner's confusion to anxiety. Id. at 440. Dr. Steinman agreed he was neutral about whether petitioner has myasthenia gravis. Id. at 441-42. He said the foreign accent was not associated with dystonia. Id. at 443. Dr. Steinman thinks petitioner was manifesting anxiety, frustration, and fear, but he does not think that makes it a conversion disorder. Id. at 444. He thinks doctors were perfectly reasonable to suggest anti-anxiety treatments. He thinks dysautonomia is at the heart of the whole matter. Id. Dr. Steinman thinks dystonia as a manifestation of dysautonomia could be due to an autoimmune disease. Id. at 445. A vaccination could cause dystonia if it were due to an autoimmune problem and if it is a manifestation of the dysautonomia. Id.

On the third day of testimony, on cross-examination, Dr. Steinman said the first time he met petitioner was at the hearing. Id. at 467. He stated that petitioner's injury is restricted to the autonomic nervous system and the vagus nerve.[161] Id. at 467. He said petitioner's case is complex and whether or not she has myasthenia is not central to his conclusions. Id. at 469. He does not think petitioner had Lambert-Eaton syndrome, which Dr. Sheean included in a differential diagnosis. Id. The undersigned asked Dr. Steinman if the undersigned held petitioner did not have myasthenia gravis, would Dr. Steinman's opinion be she still had dysautonomia and the vaccine caused everything she has, and he said that is a very accurate summary of his reasoning. Id. at 471. Dr. Steinman said myasthenia gravis is "not a cornerstone" of his thinking. Tr. at 475.

As for foreign accent syndrome, Dr. Steinman explained petitioner has bulbar weakness from involvement of her autonomic nervous system, controlling speaking and breathing. Id. at 476. She could not speak naturally unless she breathed physiologically. He also said if

[161] "Vagus nerve damage can occur following upper respiratory viral infections. These infections initially involved symptoms such as cough, nasal congestion and runny noses. Symptoms that persisted in patients identified as having post viral vagal neuropathy, or PVVN, included cough, throat clearing, difficulty speaking and vocal fatigue." What Are the Causes of Vagus Nerve Damage?, LIVESTRONG, https://www.livestrong.com/article/148586-what-are-the-causes-of-vagus-nerve-damage/ (last visited April 9, 2019).

petitioner had abnormal breathing due to mechanisms controlling her diaphragm through her vagus nerve, that could also cause foreign accent syndrome. Id. He said, "I'll say, frankly, right now, it's not the case in such a complex spectrum of behavioral abnormalities that I am even going to attempt to say that I can explain everything by point A or B." Id.

Dr. Steinman continued:

> Your very good questions force me to think, and the vagus nerve, heavily myelinated, a cornerstone of my argument is that there is molecular mimicry to myelin. She may have abnormalities of her vagus nerve that contribute to POTS and her orthostatic tachycardia. It may interfere with her respirations.

Id. at 477. Dr. Steinman then proceeded to imitate talking nasally as if he had respiratory problems. The undersigned said that he did not sound as if he comes from England. Id.

The undersigned reminded him that he linked petitioner's foreign accent syndrome to her having myasthenia gravis and asked him if it were affiliated with other conditions. Id. at 478. Dr. Steinman responded that he was not "the definitive authority on foreign accent syndrome" and could only say it would not be specific only to myasthenia gravis. Id. Dr. Steinman continued, "I'm not personally wild about myasthenia" and repeated his admiration for Dr. Sheean. Id. at 480-81.

Dr. Steinman said, "I've mentioned that there are certain aspects of this case that I thought long and hard about on do I want to come here and put my reputation on the line? And I'm here. And I feel very strongly about this case." Id. at 484-85. He repeated he is "not a world's expert on foreign accent syndrome." Id. at 486. He said it was a tough call whether petitioner has a psychiatric or organic illness or both. Id. at 487.

Dr. Steinman questioned whether the vagus nerve was central or peripheral. Id. at 491. It has a huge, wandering path. He does not make this separation. He said dystonia can affect both the autonomic and non-autonomic parts of the nervous system. He has been thinking about it as mainly a central nervous system disorder, but then the autonomic nervous system has a central input. Id. at 491. He said there are amazing linkages between the peripheral and central nervous systems. Id. at 491-92. He believes that petitioner had dystonia and it was a manifestation of dysautonomia. Id. at 492. He did not think petitioner had dystonia until he saw petitioner's weird movements going away when she walked backward in the videos. Id. at 492-93. He questioned how anyone could ever know[162] that walking backward would make her flailing go away. Id. at 493.

[162] Dr. Steinman must not have watched the videos in which petitioner's then-husband said petitioner looked up dystonia on the Mayo Clinic website and learned about sensory tricks after the Johns Hopkins physical therapist told her she had dystonia. Ex. 58, Video #409_0279_01. This was not the first time petitioner looked up a disease on the Internet. She told Dr. Mannon at Inova Fairfax Hospital on September 26, 2009 that she looked up Lyme disease on the Internet because she knew people who had it and she thought she did, too. Med. recs. Ex. 44, at 14.

Dr. Steinman said that if the vagus nerve is impaired, a person can get autoimmune diseases. Id. at 496. If you stimulate the vagus nerve, you can overcome the issues resulting with vagal nerve abnormality. He mentioned articles by Kevin Tracey which were not in evidence to support his statement. Id. Dr. Steinman thinks vagal nerve abnormality caused petitioner autoimmune dysautonomia. Id. at 497. Dr. Steinman said he could analyze whether a symptom was autonomic or non-autonomic only on a case by case basis. Id. at 499.

Dr. Steinman thinks that petitioner responded to Dr. Buttar's treatment because IV therapy improves POTS. Id. at 502. He thinks petitioner's POTS is autoimmune and part of her dysautonomia. Id. at 503. Dr. Steinman testified that he has never seen any evidence that hyperbaric therapy works for anything but diving injuries. Id. at 504. Dr. Steinman clarified his opinion by stating that POTS would not have anything to do with petitioner's ability to walk forward when she embraced her husband and said she was healed. Id. at 505. He said POTS would apply only to her cardiac physiology, but it would be part of a broader dysautonomic syndrome and the dystonia was due to an autoimmune dysautonomia. Id. When the undersigned asked Dr. Steinman why the IVs' improving her blood volume would help petitioner walk forward and speak normally, Dr. Steinman said, "I can't integrate all of these things, no. I could try, but I'm not going to." Id. at 506. He went on to explain the placebo effect:

> So, in imparting to an individual that something is going to help them, we humans are complex, we often get more of a positive response. You know, if you could put a placebo effect in a bottle and make a drug out of it, part of it is that art of practicing, and maybe Buttar and maybe this other guy, they're good salespeople, and they tell somebody they're going to be better and they are better for a while.
>
> So, there may be inconsistencies, and believe me, I saw inconsistencies in the films and in the history, not everything satisfied me 100 percent, but in sum, and in summary, not – I felt that there's clearly something organic going on, and then I made a theory.

Id. at 507.

He continued explaining the placebo effect:

> And I'm implying when a person has a positive response or even thinks they're [sic] having a positive response, there may be a ripple effect into other areas, and she may, indeed, be – you know, do better in other areas for a while.

Id. at 508.

Dr. Steinman said he considers autoimmune dysautonomia and autoimmune autonomic neuropathy as equal. He stated autoimmune autonomic neuropathy can involve either the peripheral nervous system or central nervous system or both nervous systems. Id. He considers

petitioner to have autoimmune autonomic ganglionopathy which is negative for antibody to ganglionic acetylcholine receptors. Id. at 510. He testified autoimmune autonomic ganglionopathy is subsumed in autoimmune dysautonomia. Id.

Dr. Steinman admitted that he included in a chart (Ex. 93) he created of doctors who diagnosed petitioner with autoimmune dysautonomia the name of Dr. Urrutia at Johns Hopkins University without also including Dr. Urrutia's later note that petitioner's symptoms did not fit a physiologic paradigm and, instead, might be her reaction to anxiety. Id. at 514, 515.

Dr. Steinman admitted that volume correction of petitioner's POTS from Dr. Buttar's IV treatment would not explain petitioner's positive response to Dr. Buttar's rubbing transdermal DMPS drops on her arms, and Dr. Steinman noted for the transcript of the hearing that he laughed in agreement with respondent's counsel's question ("let it be known for the record that I had to laugh in agreement"). Id. at 518.

Dr. Steinman said that the ability to walk backward and sideways but not forward is symptomatic of dystonia, but not myasthenia gravis, recognizing an error in his list of symptoms common to each disease (Ex. 94). Id. at 525, 526. In a list (Ex. 94), he said that cold spots in the back of petitioner's head were irrelevant for autoimmune dysautonomia. Id. at 527. But in a later list of symptoms of autoimmune dystonia (Ex. 175), he admitted he made a mistake when he included cold spots in the back of petitioner's head. Id. at 528. Dr. Steinman said, "And as I'm sitting here, I don't put a lot of – I can't explain, as I sit here today, what on Earth the cold spots in the back of the head was all about." Id. at 529.

Dr. Steinman admitted that his understanding was that 2009 Fluzone vaccine that petitioner received in 2009 was composed of A/California/2009, A/Victoria/2009, and B/Brisbane/2008, and could induce anti-myelin basic protein antibodies and antiganglioside antibodies. Id. at 530. He also said he based the symptomatology portion of his molecular mimicry theory on the components A/California and A/Victoria strains that he believed were in the 2009 Fluzone vaccine that petitioner received. Id. at 530-31. Respondent then introduced Exhibit WWW, which was a Sanofi-Pasteur press release stating the actual components of the 2009 Fluzone vaccine (which were not what Dr. Steinman had based his opinion on). Id. at 531, 532. The three components were A/Brisbane strain H_1N_1-like virus, A/Brisbane strain H_3N_2-like virus, and B/Brisbane strain-like virus. Id. at 532. Dr. Steinman admitted there was a discrepancy in what he conceived were the components with what were the actual components in the vaccine petitioner received. Id. He said it would require a little detective work. Id. at 534. Dr. Steinman had looked at the wrong flu season (2010-2011) for his understanding of the components of Fluzone in 2009. Id. at 535.

On redirect, Dr. Steinman said the most important mimic known is the antiganglioside. Id. at 542. All these different viruses will elicit antiganglioside independent of their differences in protein structure. Id. at 543. They have enough sites in their proteins to get glycosylated. If a hemagglutinin gets glycosylated with a ganglioside, it can elicit antiganglioside antibodies. He said many questions remain unanswered why only once in a while does epidemiology reflect

this, but he thinks it is highly likely that the 2009 vaccine petitioner received could elicit an antiganglioside antibody. Id.

Dr. Lancaster's Testimony

Dr. Eric Lancaster testified for respondent. Id. at 547. Dr. Lancaster is an assistant professor at the University of Pennsylvania, conducting research concerning the mechanisms of autoantibodies to different brain and peripheral nerve proteins, to provide clinical care for patients in his clinic which mostly focuses on people with autoimmune neurological conditions, and to assist with diagnostic testing of autoantibodies for patients with various forms of suspected autoimmune encephalitis. Id. at 548-49. He also educates residents, fellows, and medical students. Id. at 549. He has a Ph.D. in neuroscience. The topic of his thesis was the response of autonomic neurons of the vagus nerve to injury. He is board certified in neurology, neuromuscular medicine, and electrodiagnostic medicine. Id. He sees patients with various forms of autoimmune encephalitis, autoimmune neuropathies, autoimmune neuromuscular junction disorders, and antibody-mediated neurological problems. Id.

Dr. Lancaster has diagnosed and treated about 30 patients with dysautonomia. Id. at 550. He has diagnosed and treated about 30 patients with myasthenia gravis. He has seen about five patients with autoantibodies to the ganglionic acetylcholine receptor. Id. He has diagnosed and treated patients with dystonia. Id. at 551. Dr. Lancaster has done research on the response of vagal sensory neurons to injury, which is an important part of the autonomic nervous system. Id. Dr. Lancaster believes that dystonia and dysautonomia are very different and need to be considered separately. Id. at 552. Respondent offered Dr. Lancaster as an expert in neurology and autoimmune neurological disorders and diseases. Id. at 553. Petitioner had no objection. Id. at 554.

Dr. Lancaster's opinion is that petitioner's primary diagnosis is conversion disorder. Id. at 554. Dr. Lancaster explained conversion disorder is a subconscious response to stress. Id. at 555. A person with conversion disorder can manifest many abnormal medical problems for which a physiologic explanation does not exist, but subconscious stress is the explanation for the manifestations. Some of the most common manifestations of conversion disorder occur in neurology, e.g., events that look like seizures, but do not involve actual seizures in the brain, but are precipitated by stress rather than by an abnormal discharge of electricity among neurons in the brain. Id. Other manifestations might include an abnormal gait. Id. Further manifestations might be weakness for which no physiologic cause is apparent or is likely to exist. Id. at 556. Some patients can have more than one manifestation. Conversion disorder problems are relatively common in neurology. Dr. Lancaster has had about 50 to 100 patients with conversion disorder. It is not a rare problem. Id.

Dr. Lancaster said that a person manifesting conversion disorder generally is not conscious of what is causing his or her symptoms. Id. at 557. Dr. Lancaster distinguished between someone with conversion disorder and a malingerer. A malingerer consciously decides to fake a medical illness with a concrete goal. For example, someone who pretends to have a cold to call in sick in order to go out and play golf is a malingerer. Conversion disorder is

subconscious. There is no apparent gain for the patient. In many cases, what the person with conversion disorder is doing makes his or her life difficult, more unpleasant, and could eventually hurt him or her. Id.

Dr. Lancaster stated that a neurologist, rather than a psychiatrist, more often diagnoses conversion disorder. Id. at 558. He stressed the extreme importance of excluding physiological causes. He stated specific clues often suggest the diagnosis of conversion disorder. Id.

Dr. Lancaster testified that petitioner had a variety of symptoms in the first several months after her supposed seizures that in totality do not admit to an organic or physiologic cause. Id. at 559. She had many different manifestations. Some that were most prominent and aid in diagnosing her with conversion disorder include repeated attacks of severe weakness resulting in apparently profound inability to move, followed by a rapid recovery. Other events included the unusual nature of her walking problem with a wildly lurching gait that made petitioner seem profoundly off balance and yet required her to have a great deal of balance to perform. Dr. Lancaster said this manifestation is called astasia-abasia, which is seen in the videos. Id.

Another manifestation of petitioner's conversion disorder is her speech problem which was inconsistent with an organic etiology, in Dr. Lancaster's opinion. Id. at 560. Her speech problem fluctuated among four or five different presentations, as apparent on some of the videos.

Dr. Lancaster said another manifestation of petitioner's conversion disorder was the nature of her responses to some of her tests, e.g., her first tilt-table test. Id.

In sum, Dr. Lancaster could not give any physiologic explanation for the great majority of petitioner's most serious and impairing symptoms in the first three months or so after the onset of her condition and concludes she had conversion disorder. Id. The objective testing at Johns Hopkins and other centers was extremely helpful in ruling out an organic etiology. She had normal brain MRIs, normal nerve conduction studies and EMGs on several occasions, normal lumbar puncture result, and normal EEGs. Id. Her tilt-table testing was also extremely helpful because she had profound and impairing symptoms when tilted, but her blood pressure remained normal. Id. at 560-61. In other words, she did not have hypotension as the cause of her symptoms. Id. at 561.

Dr. Lancaster said that the physicians who were directly observing petitioner and maintained medical records in the first three months after the onset of her condition often suspected a psychiatric etiology. Many of them saw her gait and thought it did not have an organic basis or observed other symptoms which they wrote did not have an organic basis. The later records of physicians who did not observe petitioner early may not have had all the information. Id. The video evidence was extremely helpful. Id. Normally. Dr. Lancaster said he would spend an hour with a patient in the office to diagnose him or her with conversion disorder. Id. at 562. With the videos of petitioner, Dr. Lancaster said he was able to observe petitioner's behavior directly over a long period of time, i.e., many hours. He was able to stop, go back, view something again, look at it in detail, observe petitioner's gait, speech, and other

symptoms. Dr. Lancaster said petitioner is the person with conversion disorder that he has been able to study in the most detail because of the videos. Id.

Dr. Lancaster stated that the videos show petitioner having very rapid changes in her level of strength, in her apparent level of consciousness, with wild shaking-like events taking several forms, shaking limbs at rest, shaking limbs with movement, shaking of her head, and forward thrusting of her head. They also show multiple changes in her speech, sometimes speaking with a "flat out" British accent or close to it. Id. They show that sometimes she spoke in a slow and exaggerated way that was harsh and strained. Sometimes she spoke very quietly and whispered. Sometimes she shifted rapidly from one to another. Id. at 562. He thinks there were at least five distinct changes. Id. at 562-63. He said, "The fact that there were just so many different manifestations of the dysarthria weighs pretty heavily against an organic cause." Tr. at 563.

As for petitioner's gait, the videos show her walking in the characteristic way of astasia-abasia where she appears wildly off balance, lurching forward, jerking violently as she is moving, and yet not falling down, maintaining excellent balance. Id. Dr. Lancaster said it takes balance and strength to perform that. Someone who was off balance or uncoordinated could not perform that and would fall down. Id.

Dr. Lancaster said he observed events that were highly characteristic of nonepileptic spells, sometimes call nonepileptic seizures which are not really seizures but rather psychiatric seizures. For instance, petitioner would appear to have profoundly altered consciousness and yet still respond to people. She would still be able to move both her arms during an attack. She would close her eyes and shake her extremities during an attack. Id.

Dr. Lancaster also called attention to the profound effect of suggestion in the videos. Id. at 564. He said Dr. Buttar gave petitioner "an incredibly powerful treatment" in his office, i.e., suggestion. Dr. Lancaster stated Dr. Buttar in the videos would tell petitioner that something was going to work and help her and, in agreeing with Dr. Steinman, Dr. Lancaster said there was "an incredibly powerful placebo effect" to which petitioner had a great response. Dr. Lancaster said that it did not seem to matter what the exact physical treatment was. But any time someone suggested the treatment would work for petitioner, it did profoundly, at least transiently. Id. Dr. Lancaster said he did see in the videos that petitioner had tremors, which he would describe as psychogenic tremors. Id.

Dr. Lancaster's opinion is that the August 23, 2009 flu vaccination is highly unlikely to have resulted in an adverse reaction. Id. His reason is that conversion disorder is a manifestation of stress and flu vaccine does not cause it. Id. at 565.

Dr. Lancaster described the autonomic nervous system. It refers to the very specific parts of the nervous system that handle a small subset of neuronal functions that are completely outside conscious control. These specific functions include regulating the following: blood pressure; heart rate; sweating; salivation; tearing; and the size of the pupil. Id. The autonomous nervous system is sometimes divided into components, such as the sympathetic nervous system (controlling mostly sweating, cardiovascular responses, dilating of the pupils), and the

parasympathetic nervous system (controlling salivation, moving of the intestinal tract, slowing down the heart rate, constricting the pupil, and assisting with urination). Id. at 565-66.

Sometimes, doctors include the enteric nervous system as part of the autonomic nervous system. Id. at 566. The gut itself has as many neurons as the spinal cord in order to allow the gut to squeeze properly and coordinate its motions. Thus, the enteric nervous system constitutes the third main component of the autonomic nervous system.

Dr. Lancaster said the autonomic nervous system is mostly in the periphery, involving nerves running out to the heart, glands, blood vessels, skin (to control sweating and goose flesh). He stated the autonomic nervous system also has a central component that is a very small part of the central nervous system, involving monitoring and controlling these bases on a central basis. Id. For example, the light pathways, when a light shining in one's eye constricts the pupil, is part of the autonomic nervous system. Id. at 566-67. The central pathways for regulating one's temperature and determining whether someone should be sweating or shivering is considered part of the autonomic nervous system. Id. at 567.

Dr. Lancaster stated it is important to distinguish what the autonomic nervous system does not do, such as voluntary motion which voluntary motor control performs from the brain through the spinal cord to the peripheral nerves to muscle. Walking involves the activation of muscles from the nerves directing those muscles to work and is not based in the autonomic nervous system. Id. He noted that "autonomic" does not mean "unconscious." Id. Dr. Lancaster said that speech, understanding speech, visual perception, and hearing are completely outside voluntary control but are not considered part of the autonomic nervous system. Id. at 568. Balance is generally outside voluntary control and is not considered part of the autonomic nervous system. The perception of light and sound are not considered part of the autonomic nervous system. Thus, there are small parts of the central nervous system that are autonomic. Id. There are also parts of the peripheral nervous system that are considered autonomic that do very specific things. Id. at 569.

The undersigned asked Dr. Lancaster what the vagus nerve is. He replied the vagus nerve comes out of the brainstem, travels a long, winding course, and touches many inner organs. The vagus nerve is mostly autonomic and considered part of the parasympathetic system. Its autonomic functions include slowing the heart rate and balancing out the sympathetic tone that could increase heart rate. It helps the intestines move food along and tells the intestines to digest faster when someone puts food in his or her mouth and into his or her stomach. It mediates the activity of the liver with glucose control. It also has some role in sensing blood pressure. Id.

Dr. Lancaster disagrees with Dr. Steinman that the vagus nerve controls breathing. Id. at 569-70. Dr. Lancaster said the phrenic nerve which is under voluntary control and not part of the autonomic nervous system controls breathing. Id. at 570. He said the vagus nerve also has some components that are not autonomic.

Dr. Lancaster said a long QT is not autonomic. Id. at 572-73. The QT refers to the fact that, as the heart contracts, it conducts electrical signal across itself through the heart muscle

cells. Id. at 573. A long QT means that one part of that conduction pathway is too slow. That is a process with the heart muscle cells that conduct the electricity, not with any nerve cell. When someone has a long QT, he or she has a problem with the conduction of certain muscle cells within the heart that are supposedly specialized to conduct the impulse rapidly through the heart. In order for some disease to cause a long QT, there must be a mechanism for that disease to affect heart muscle cells, not nerve cells. Dr. Lancaster said that there is nothing anyone can do to a nervous system to give someone a long QT syndrome. It is a cardiac conduction phenomenon. Its cause can be genetic, drugs, or toxins. Id. Dr. Lancaster said he does not know why petitioner has long QT syndrome. Id. at 574. He does not think she has dysautonomia per se. The long QT syndrome could be from any of the number of medications petitioner is on. Perhaps she developed it over time. Id.

Returning to digestion, Dr. Lancaster said that moving of the tongue, chewing, and initial swallowing are not autonomic. They are under voluntary control. Salivation and drooling are autonomic. Id. Beyond the initial voluntary initiation of swallowing, from the lower two-thirds of the esophagus on down is considered autonomic. Id. at 575.

Speaking is not a function of the autonomic nervous system. The vocal cords, the tongue, and the mouth are under voluntary control. Dr. Lancaster's opinion is that petitioner does not have myasthenia gravis.

Dr. Lancaster said his definition of the autonomic nervous system is widely accepted in the neurologic community and would come straight out of any neurology textbook or any prominent review of the subject. Id. For example, orthostatic hypotension means one's system for maintaining blood pressure is not working right and becomes stressed when one stands up. Id. at 577. When one stands up, one becomes dizzy and might pass out or become lightheaded. If one lay down again, the symptoms would rapidly resolve. Someone could make too much saliva (drool) or too little saliva. Id. Someone with autonomic nervous system disease might have trouble squeezing his or her bladder out or allowing the bladder to relax and accommodate fluid. Id. at 577-78.

Dr. Lancaster said cognition is not considered an autonomic function. Id. at 578. The only connection between cognition and autonomic function is the maintenance of blood pressure which enables someone to think. If someone stands up and cannot maintain his or her blood pressure, he or she will feel dizzy and pass out. Id. Someone's consciousness, thoughts, emotions, feelings, understanding, and memory are not autonomic, and the autonomic nervous system does not directly control them. Id. at 579. The medical community does not consider the autonomic nervous system as the mediator of dystonia. Dystonia is an abnormality of motor control involving areas in the brain that control and execute movements. Nothing in the peripheral nervous system causes dystonia. It is a problem of voluntary muscles. It is a problem of the central nervous system. Id.

Dr. Lancaster said the most common form of dystonia is writer's cramp in which, while someone is writing, the motion triggers hyperexcitability in certain motor pathways, generally in the brain, and the hand locks into an uncomfortable position for a period of time. Id. at 579-80.

He stated another form of dystonia might involve the leg as someone is walking. Id. at 580. The leg might have abnormal tone, and the leg would be turned in and extended at the ankle. Vocal dystonia involves having a fixed, hoarse, or strained voice. Motion can trigger some dystonia while other dystonia occurs constantly whether the person is moving or not. Some dystonia can activate part of the sympathetic nervous system rarely when a hand might feel clammy and cold while contracted. That would be very unusual. The overwhelming manifestation of dystonia is an abnormality of the voluntary motor control system, not the autonomic nervous system. Id. Dr. Lancaster testified that the Marsden and Fahn articles describing torsion dystonia in children and sometimes adults, involving walking backward while unable to walk forward, are describing a central nervous system condition. Id. at 580-81. Dr. Lancaster said that the overwhelming majority of neurologists would be shocked and surprised to hear anyone attributing a complex motor coordination problem to dysautonomia, rather than the central nervous system. Id. at 580.

Dr. Lancaster then discussed the Marsden article which he said is an excellent description of patients with dystonia involving many classic features. Id. at 582. Dr. Lancaster thinks the patients in the article most likely had a genetic disease, not an autoimmune disease. Those patients sometimes have complex gait disorders, but not astasia-abasia. Dr. Lancaster also noted that once these patients had onset of dystonia, their dystonia tended to be the same for years. Their manifestations of dystonia are relatively static or worsen over time. They do not have one kind of wild lurching movement one day and then it is all gone and then a completely different wild movement the next day, and yet another movement the third day, and then it is all better. Id. Dr. Lancaster said that IVIG would be completely useless to treat patients with dystonia because it is not autoimmune. Id. at 583.

Dr. Lancaster said there are very rare autoimmune diseases that might manifest dystonia for which he would use immune therapy. For instance, someone with anti-NMDA receptor encephalitis might have dystonic posturing as part of that brain disease. Id.

Dr. Lancaster said that Fahn in Ex. 129 described the clinical variance of idiopathic torsion dystonia. Id. at 584. Since the publication of Fahn's article, some of these unknown ("idiopathic") causes have turned out to be genetic. Dr. Lancaster does not believe the neurology community believes that idiopathic torsion dystonia is an autoimmune disease. It would be very surprising to treat such a patient with IVIG or steroids. Id.

Dr. Lancaster explained why notable treating doctors such as Dr. Grubb, Dr. Sheean, and Dr. Atiga diagnosed petitioner with either dysautonomia or myasthenia gravis because they did not have the privilege of observing all of petitioner's florid symptoms of conversion disorder early on in 2009. Id. at 585. By the time, in 2012 or 2013, petitioner saw these doctors, the medical records mention the gait disorder much less often or not at all during a visit. Id. at 585-86. Her abnormal movements and seizure-like events were mostly gone. Id. at 586. Dr. Lancaster contrasted these doctors with Dr. Urrutia who saw petitioner early on and suspected a psychogenic etiology. Id.

Moreover, Dr. Lancaster explained that the later doctors were working with limited information and would write down petitioner's history that she had a positive tilt-table test,

which they would accept at face value. Id. But, Dr. Lancaster continued, if they had read the original tilt-table report, it is unclear if it concluded petitioner had autonomic dysfunction. Id. What happened in the first tilt-table test is that they tilted petitioner to an upright position and she had a long period of normal blood pressure and normal pulse, while at the same time, she manifested florid symptoms of abnormal speech and feeling profoundly unwell. Id. at 587. That is not an autonomic problem. That was not a blood pressure regulation problem. The mechanism of blood pressure problems is dropping the blood pressure. If petitioner had an autonomic disorder, when she was upright after being tilted, her blood pressure would drop, her brain would not be well-profused, she would feel lightheaded and unwell, and perhaps she would pass out. Id. Dr. Lancaster thinks in many of the later doctors' cases, they were working with incomplete information.

As for petitioner's gastrointestinal concerns, she had a number of normal studies early on. Id. Dr. Frisca Yan-Go, an expert autonomic neurologist, reviewed all petitioner's tests and concluded petitioner did not have significant autonomic disorder. Later on, petitioner had one abnormal gastric emptying test result while she was taking Sandostatin, which can cause that result on the test. Id.

As for petitioner's having myasthenia gravis, that is a difficult diagnosis. Id. at 588. Petitioner had patterns of fluctuating weakness. Ninety percent of patients with myasthenia gravis have antibodies to acetylcholine receptor and MuSK. Petitioner did not. Then she was tested with the repetitive stimulation test and that was normal. But ninety percent of myasthenia gravis patients have an abnormal result. Dr. Lancaster asked would one or two percent of myasthenia gravis patients have both a negative antibody to acetylcholine receptor and MuSK and negative repetitive stimulation test. He concluded that the myasthenia gravis diagnosis of petitioner did not have much in the way of actual factual basis to support it. Then, looking back at petitioner's episodes of profound weakness that instantly got better, episodes of slurred speech that got better, Dr. Lancaster thinks what was actually happening was ongoing conversion disorder. The more florid manifestations of petitioner's conversion disorder became much more subtle over time. Id.

Dr. Lancaster noted that petitioner's antibody tests were done both before and after she went on IVIG. Id. at 589. They were widely separated in time. He does not think IVIG would make the antibody test negative. Patients with myasthenia remain antibody positive for years and years, even when they go into clinical remission. Those who are antibody negative do not have myasthenia any more. Id.

Dr. Lancaster said petitioner took a second tilt-table test. Id. at 590. The first tilt-table test was July 15, 2010 (Ex. 23, at 1,2) and the second one was on September 9, 2010 (Ex. 37). Id. at 590-91. The second tilt-table test was done at Ohio State University and the findings were normal but for variable blood pressure and pulse pressure during 15 minutes of orthostatic stress. Id. at 591. Heart rate increased by 35 beats per minute to an absolute maximum of 123 beats per minute. Petitioner had normal heart rate variability in response to deep breathing. Ohio State's conclusion was that petitioner's tilt-table results were consistent with grade two orthostatic intolerance, POTS, a conclusion with which Dr. Lancaster disagrees. Id.

Dr. Lancaster referred to the Plash article (Ex. E), which is a study of different diagnostic criteria using tilt-table testing to diagnose patients positive or negative for POTS. Id. at 592. Dr. Lancaster said the key question is for what length of time should technicians tilt their patients and how high should a patient's pulse go in order to differentiate normal patients from those with POTS. He described the Plash article as trying a number of different conditions. Id. at 592. He said another important component was whether to use standing or tilting as the more likely determinant of who is normal and who has POTS. Id. at 592-93. Dr. Lancaster stated the question is whether to use an increase of 30 or 37 beats per minute and whether to tilt the patient up for 10 minutes and keep observing the patient for that increase or to tilt the patient up for 30 minutes and observe the patient for all that time to see if there is an increase. Id. at 593. The Plash article states that a 10-minute tilt correctly identified 93 percent of POTS patients, but also identified 60 percent of normal control subjects as having orthostatic tachycardia, which was a false positive. Id. This suggests that tilting for 10 minutes, 30 beats per minute, is highly sensitive but has poor specificity, that is, it classifies normal people as positive. Id. at 593-94. The Plash authors found that increasing the heart rate cutoff between POTS and normal to 37 beats per minute resulted in a more specific test which had good sensitivity. Id. at 594.

As for the second tilt-table test petitioner took at the University of Ohio (Ex. 37). Dr. Lancaster said that petitioner had an increase of heart rate by 35 beats per minute at 15 minutes into the test. Id. He noted that between 10 and 30 minutes, from 40 to 80 percent of normal people have the same result. Id. Dr. Lancaster stated this is an extremely non-specific finding that a great number of normal people have. Id. at 594-95. He said that petitioner's second tilt-table test was not "gold-plated" evidence that she has an autonomic disorder. Id. at 596. He does not think the results of this test diagnose petitioner with POTS or autonomic dysfunction.

Dr. Lancaster then looked at the first tilt-table test done July 15, 2010, which was 10 months after vaccination. Id. The test results were normal heart rate and blood pressure responses to upright tilt-table test. Id. Provocation with nitroglycerin reproduced and worsened petitioner's symptoms, which the technicians thought indicated petitioner had a form a cerebral syncope in which she had dysregulation of cerebral blood flow when upright, especially with orthostatic stress. Id. at 596-97. Dr. Lancaster disagreed with this assessment. He thought the results weighed against petitioner having an autonomic disorder because petitioner was able to regulate her blood pressure during the test. Id. at 597. He does not think that focal regulation of cerebral blood flow when upright is a well-established disease. Id. He does not know why the technicians gave petitioner nitroglycerin, which is a vasodilator. Id. at 598. Dr. Lancaster does not agree with the cerebral diagnosis and thinks it relates to the issue of the PET scan, where the technician thought there was a focal abnormality in distributing blood around her brain. Id. at 598-99. He thinks the technician got one abnormal SPECT scan to support that diagnosis, which Dr. Lancaster noted Dr. Steinman was skeptical about and Dr. Lancaster entirely agrees that those tests are very non-specific. Id. at 599. Petitioner had a subsequent PET scan of her brain that did not show any abnormality at all. Id.

As for petitioner's doctors diagnosing her with illnesses she did not have, Dr. Lancaster explained they were recording what petitioner told them, oftentimes a diagnosis outside their

specialty which they did not personally check out or create for themselves. Tr. at 601. For instance, Dr. Atiga, a cardiologist, repeated petitioner's diagnosis of dystonia even though he is not a neurologist and did not check that petitioner had dystonia. Id.

Dr. Lancaster testified that dysautonomia does not impact cognition. Id. He said that dysautonomia does not impact a person's ability to speak or pronounce words. Id. at 601-02. Dr. Lancaster also stated that dysautonomia does not impact someone's gait. Id. at 602. If someone did not have sufficient blood pressure, he or she might get lightheaded or fall down, but not otherwise have an abnormal gait. There is a form of dysautonomia that is autoimmune, which is autoimmune autonomic neuropathy sometimes called autoimmune autonomic ganglionopathy. In most cases, it is a relatively acute illness with a timing similar to that of GBS over several weeks or months with a profound failure of the autonomic nervous system. Id. Patients with autoimmune autonomic neuropathy manifest pupils unresponsive to light which might be completely fixed. Id. at 603. They are unable to salivate. They have profound inability to maintain blood pressure. If you give them a tilt test, their blood pressure drops straight down to 40/20 and they are out. For people with autoimmune autonomic neuropathy, their heart rates are fixed like a machine and do not vary. They have profound abnormalities in sweating. Huge parts of their bodies are unable to generate any sweat. They might have profound gastroparesis such that if they put food in their stomach, nothing happens. Autonomic autoimmune neuropathy is not subtle. They have sexual dysfunction and urinary dysfunction. Id.

Dr. Lancaster stated that half these patients have an antibody to the form of the acetylcholine receptor expressed almost entirely in the autonomic nervous system, called alpha 3 acetylcholine receptor. Id. at 604. Dr. Lancaster said that petitioner does not have this clinical picture. Her medical records do not document any significant problem with her pupils, any failure of salivation, any failure of sweating, or urinary or sexual dysfunction. Her cardiovascular tests do not show any significant abnormality in maintaining her blood pressure in response to several stresses. The fact that she could run an 8K race near the peak of her symptoms indicates her excellent cardiovascular sympathetic function. When someone runs, he or she has to increase his or her cardiac output, i.e., the amount of blood being pumped by the heart, by five or six times above normal. Id. It goes to 500 percent of normal. Id. at 604-05. Someone running has to make the heart beat about three times faster, squeeze about twice as much blood in each squeeze, and control the tone in all the other major arteries of his or her body to distribute it to the most active muscles and constrict other blood vessels to maintain the tone so that the quadriceps muscles get their fair share and keep it from getting to the brain and kidneys. Id. at 605. Dr. Lancaster said maintaining blood pressure during an intense prolonged run is an "amazing feat of the autonomic nervous system." He notes petitioner did it perfectly. Id.

Diagnosing autoimmune autonomic gangliopathy would include the most useful test, i.e., the test for ganglionic acetylcholine receptor antibody. Id. If a patient does not have that antibody, Dr. Lancaster said he would not be totally sure what that patient had. Id. at 606.

Dr. Lancaster said that dysautonomia is not a variant of GBS. Id. at 607. In patients with GBS, the predominant symptoms are in the non-autonomic parts of the peripheral nervous system including demyelination of the nerves that allow someone to move his or her muscles and to feel sensations. Id. Some patients with GBS also have significant effects on their autonomic nervous system. Id. at 607-08. Dr. Lancaster said he does not know how anyone could diagnose someone with GBS who has an isolated autonomic syndrome. Id. at 608. It does not make sense to him and he does not think most neurologists would accept it. Patients with GBS who have autonomic failure have overwhelming evidence of weakness and numbness in the non-autonomic parts of their nervous system. The weakness and numbness tend to peak at the same time as the autonomic failure rapidly improves when the person gets better. Id.

Dr. Lancaster said he disagrees with Dr. Steinman on several levels. He said petitioner has conversion disorder, not an autonomic disorder, not autoimmune autonomic neuropathy, and not some sort of autonomic-only version of GBS. Id.

Dr. Lancaster defined dystonia as a complex and abnormal contraction of certain muscle groups, either with motion or at rest. Id. at 609. The brain primarily mediates dystonia, particularly in the areas for planning complex motor movements called the motor cortex, the pre-motor area, and the supplemental motor areas. Other structures such as the basal ganglia and the cerebellum help coordinate movements, and none is part of the autonomic central nervous system. Id.

Dr. Lancaster distinguished dystonia from dysautonomia. Id. at 610. Dystonia is a very complex motor problem emanating from the brain that does not involve the autonomic nervous system in any substantial way. Dysautonomia involves failure of other autonomic functions but does not involve dystonia. Id. Dr. Lancaster discussed the Suarez paper (Ex. 132), whose senior author is Dr. Philip Low, an esteemed expert on autonomic disorders. The paper discusses idiopathic autonomic neuropathies without any known cause. Id. at 610-11. Dystonia is not included in the paper. Id. at 611. The paper does discuss the symptoms of autoimmune autonomic neuropathy that Dr. Lancaster mentioned in his testimony: pseudomotor testing (sweating) and adrenergic function testing (blood pressure, cardiovascular functions). But the authors do not discuss dystonia. Id.

Dr. Lancaster defined myasthenia gravis as a disorder of acquired muscle weakness occurring insidiously over weeks to months. Id. The pattern of the weakness in myasthenia often centers on the muscles of the face, neck, and eyes with some involvement of the neck and shoulder girdle, but very little involvement with the smaller muscles in the hands, feet, and legs. Id. at 611-12. Some of the more characteristic aspects of patients with myasthenia gravis is that the muscles keeping their eyes open are weak, resulting in ptosis. Id. at 612. In addition, many myasthenia patients have weakness of the muscles moving the eyes. They frequently complain that they see two images because their eyes are not aligned any more. Nystagmus would be uncommon. Id. Nystagmus is a repetitive beating movement of the eyes. Id. at 613. In all of petitioner's medical records, Dr. Lancaster testified that he did not see any description of petitioner having ocular weakness. People with myasthenia gravis become objectively weak with repeated muscle use. Id. People with myasthenia gravis become weaker toward the

evening. Id. at 614. Myasthenia gravis does not fluctuate second to second or minute to minute, Dr. Lancaster said. People with myasthenia develop respiratory weakness because the phrenic nerve is not transmitting well to the diaphragm's neuromuscular junction and the diaphragm becomes weak. As myasthenia patients lose respiratory strength, they go into myasthenia crisis. Id. They become weaker over several weeks or days and each breath is labored and short. Id. at 614-15. These patients are incredibly ill and in danger of imminent death. Id. at 615. They need to go to the hospital and often to go on a ventilator. That crisis does not rapidly turn on and off from minute to minute and second to second. They can be gradually brought out of crisis over a course of days to a couple of weeks. Id.

The undersigned asked Dr. Lancaster about petitioner's episode of stridor during the hearing the day before and his medical analysis of what he saw and heard. Id. at 615-16. He responded:

> I heard the loud stridors kind of breathing sound. Clearly there was a component of stress triggering a physical illness, which is exactly what we've been talking about here. There could well be some underlying physiologic contribution to the stridor as well. For instance, she could have gastric reflux coming back up and irritating the vocal cords, and then she just in the setting of the stress, that was enough to push her over the edge. She could, as far as I know, have allergies or some other problem with her larynx that I would have no way of knowing about that until they do a full evaluation. I did not think I saw any stigmata of myasthenia at that time whatsoever. It's particularly notable that Dr. Steinman, who was close to her, kept pointing out that she's moving air well, which is the main problem with myasthenia gravis is your diaphragm is weak and you can't move air well. What she was doing probably required something like five or ten times as much respiratory effort as a normal person would have at rest, and so we observed her doing that. You could hear the very loud quality of the sound. So, I don't think that that was myasthenia gravis. There was stridor.

Id. at 616-17.

Dr. Lancaster said the normal treatment for stridor would be epinephrine to activate the sympathetic system which reduces airway inflammation. Id. at 617-18. He would probably not use Mestinon as the first treatment for stridor because it causes drooling and diarrhea. Id. at 618. But Dr. Lancaster stated Mestinon might have an "incredibly powerful placebo effect" if petitioner thinks that it would help her. Id. Dr. Lancaster said that if he were in the ER, he would not be sure what he would do and might well just give her what she thinks would help her for the placebo effect. Id.

Dr. Lancaster recalled that petitioner also went to the hospital for stridor in November 2013 when her dog and cat were fighting. Id. at 619. (Subsequent to the hearing, once petitioner filed the Medstar Georgetown University Hospital records, Dr. Gee told the Georgetown doctors that petitioner had numerous visits to hospital emergency rooms for stridor.) Dr. Lancaster said that he did not observe petitioner have an increased effort of breathing or respiratory distress the day before. Id. at 621.

Dr. Lancaster said her running an 8K race is completely inconsistent with having significant symptoms of myasthenia gravis. Her very rapid fluctuations and reports of weakness are not myasthenia. When she reported that her tongue was vocally paralyzed, that was not myasthenia. The nature of her voice and her sound during the videos when she made exaggerated increased movements in her face when speaking is the opposite of what a myasthenia patient would do. Id. At times, petitioner would whisper and, at other times, she would speak very loudly. Id. at 622. When Dr. Lancaster saw that on video, it did not look like actual myasthenic shortness of breath.

Dr. Lancaster explained that myasthenia gravis is a disorder of the neuromuscular junction, particularly of the post-synaptic neuromuscular junction. In order to move, a nerve cell that comes from your brain or your spinal cord (the central nervous system) has to go out and tell a muscle to contract by releasing acetylcholine on the muscle in a specific area called the synapse. Id. The muscle cell has acetylcholine receptors to pick up the signal and those receptors will trigger the electrical signal to the muscle. Id. at 623. If someone has an antibody to those proteins that are on the muscle at the acetylcholine receptors, that makes the connection unreliable. Then, the muscle fails and the person becomes weak. He noted that an antibody to the acetylcholine receptor in the post-synaptic membrane is a completely different disease than an antibody to nerve on the pre-synaptic membrane, which is Lambert-Eaton Syndrome. Id. Dr. Lancaster said he agreed completely with Dr. Steinman that petitioner does not have Lambert-Eaton Syndrome. Id. at 624. Dr. Lancaster said that myasthenia gravis is not some sort of vaguely defined autoimmunity to the autonomic nervous system or even to the somatic (non-autonomic) nervous system. Myasthenia involves patients with antibodies to specific proteins on muscle cells at the neuromuscular junction. Id.

Dr. Lancaster said that petitioner's allegations would encompass autoimmunity all over the nervous system, without objective evidence of damage on MRIs, EEGs, and lumbar punctures. Id. at 625. He explained this is why he is strongly of the opinion that petitioner does not have an organic etiology for her symptoms. Id. at 626. He said:

> If the symptoms were very brief, confined to one specific area, then sure, easier to come up with an organic etiology, but prolonged over a long period of time, so many different areas, so severe, that's what makes it impossible.

Id. Because petitioner did not have acetylcholine receptor antibodies and MuSK antibodies, and she had negative results of repetitive stimulation studies (she was tested twice before and after IVIG), Dr. Lancaster opined it is far more likely that petitioner does not have myasthenia gravis.

Id. at 626, 627. He noted that myasthenia gravis does not impact cognition. Id. at 627. He also noted that patients with myasthenia gravis do not have tremors. Id. at 628. He said that is actually unusual, particularly since large amplitude, violent tremors take a lot of energy. Violent tremors or shaking is not part of myasthenia gravis. He does not know of any evidence that flu vaccine causes myasthenia gravis. Id.

Dr. Lancaster then discussed the videos filed into evidence. Id. at 631. Respondent's counsel showed Exhibit AAA which was a 20/20 news story broadcast in July 2010 but showing footage from the time period of the fall of 2009 after petitioner received flu vaccine on August 23, 2009. Dr. Lancaster said one of the most prominent symptoms was language which was variable on the video. She appeared to sound Australian but then more like an English person. There was a period where she had severe stuttering. She seemed to use her facial muscles excessively while talking. Id. In addition, she had unusual movements when she was running. Id. at 632. She seemed to have great difficulty walking forward, but could walk backward or sideways, and she could run forward. Dr. Lancaster termed this astasia-abasia. Petitioner was deeply bent and shaking her body quite violently, keeping her feet on a narrow basis, one close to and in front of the other, rather than widening out her gait, yet she did not fall. Id.

The video also showed petitioner running for almost an hour without lightheadedness or the inability to maintain her blood pressure. The video showed petitioner reporting numbness. She also reported cognitive difficulties such as not thinking clearly and not being able to access her memories despite no obvious physical manifestations at that moment. The video showed her having several kinds of tremors. She appeared to have a shaking event with decreased responsiveness. She reported respiratory distress but did not appear on the video to be in respiratory distress. Dr. Lancaster said he had never seen a distressed myasthenic patient look like that. Id.

Dr. Lancaster found it very interesting that petitioner reported reading about the fact that some patients could run and not walk. Id. at 633. She read about them walking backward but not forward, and then she would go and experiment, and each time it worked. Dr. Lancaster stated, "I believe in that case each of these was an example of suggestion." Id. He explained that it worked for her because she thought it should or might work. He said the videos show this power of suggestion in other situations, i.e., the infusions, the chelation, the hyperbaric oxygen therapy, the drops on her forearms. Id. Dr. Lancaster stated, "When someone with a disease like conversion disorder thinks that something is going to work, it's very likely to work." Id. at 633-34. This is unlike myasthenia gravis where no matter how many suggestions a doctor might make, it will not make any significant difference in the patient's symptoms. Id. at 634.

The undersigned asked Dr. Lancaster if he saw on the videos dystonia or dysautonomia and he replied in the negative. He thought it interesting that petitioner referred to sensory tricks, i.e., touching her chin or jaw if she had vocal dystonia, or putting a little stimulus on her neck if she had neck dystonia. He said, "Here, again, I noticed that each of these things can occur in some patients, she goes and reads about it, and in short order it happens." Id. Dr. Lancaster stated it would be very unusual for patient to have this many different types of dystonia all at

once in which all the tricks worked perfectly, and which seem to come and go depending on what other things she was doing during the video. Id. at 634-35.

Dr. Lancaster said that petitioner's seeming to be barely able to breathe when she arrived at Dr. Buttar's clinic on October 18, 2009 was extremely unlikely. Id. at 635. As for petitioner's stuttering, compared to someone with myasthenia gravis, Dr. Lancaster said stuttering comes from the brain not from the neuromuscular junction. Id. at 635-36. Similarly, an accent comes from the brain. Id. at 636. Language is a learned process. Sometimes a brain injury, e.g., a stroke, causes someone to shift from one accent to another. More common is someone who grew up speaking a different language and switches to another language might, after a stroke, have relatively preserved parts of his brain that could still speak the original language but not the second language. Id. Dr. Lancaster never saw on video that petitioner had facial weakness, another distinguishing feature in myasthenia patients. Id. at 637.

Dr. Lancaster noted that petitioner on other videos complained of abnormal movements when she moved her tongue. He said that would be very uncommon for an organic disease. Moving the tongue would not trigger a seizure-like event if petitioner had a seizure disorder. Id.

Dr. Lancaster explained that if the autonomic nervous system is objectively impaired, it cannot function, and sensory tricks would not work. Id. at 638. He defined astasia-abasia as the characteristic gait of many psychogenic movement disorders. Id. at 639. The patient puts one foot in front of the other, bending and lurching wildly, reporting a feeling of incredible unbalance and incredible instability, yet does not actually lose balance. It is very distinctive because if someone has a problem with his or her nerves or all his or her muscles, as in peripheral neuropathy, he or she cannot feel his or her feet well. The natural response would be to stand up straight, widen out his or her gait, and move slowly with no extra movements. If a patient with a nerve or muscle disorder tried to walk as someone with astasia-abasis does, that person would fall flat on his or her face. When someone bends his or her knees deeply and puts one foot right in front of the other, that effort requires the most strength, energy, and balance. Id.

With reference to petitioner's inappropriate episodes of uncontrollable laughter, Dr. Lancaster said that the autonomic nervous system does not control laughter. Id. at 640. He added that inappropriate episodes of uncontrollable laughter would not be a symptom of dysautonomia, myasthenia gravis, or dystonia. Id. Identifying areas of the nervous system that could be invoked in petitioner's case if she had an organic problem, Dr. Lancaster said that shakings and tremor concern motor control pathways; stuttering concerns central motor control pathways; and trouble walking concerns motor pathways in the brain through the spinal cord, sensory motor fibers in the legs and muscles, and the neuromuscular junction. Id. at 641. Trouble concentrating is not attributable to the autonomic nervous system either. Id. at 642.

If petitioner did have damage to all these nervous systems, he would expect abnormal brain MRI, lumbar puncture, and other studies. Damage affecting many areas of the nervous system should leave objective evidence showing up on examinations. Id. Dr. Lancaster said that petitioner does not have any objective evidence of damage to her nervous system. Id. at 643.

Dr. Lancaster discussed the difference between someone with a dystonic gait and someone with astasia-abasia. Someone with a dystonic gait develops it gradually. Typically, his or her leg will have abnormally increased muscle tone extended at the knee and extending and turning in at the ankle. It looks somewhat like the gait of someone who had a stroke affecting a leg. It is fixed and stays the same for years. It looks the same when the person walks. The person with a dystonic gait does not lurch or shake. Someone with a dystonic gait might have trouble walking forward, but she would also not be able to walk backward. Id. Astasia-abasia, on the other hand, involves a wild gait with deeply bent knees and wild lurching movements, giving the appearance that the person is incredibly off-balance without falling. Id. at 644. If the person does fall, he or she tends to fall on a couch, on a bed, or where people can catch him or her. Astasia-abasia varies a lot from moment to moment. It is distractible. It comes and goes. It is inconsistent. Id.

Besides the lack of objective proof of petitioner's having an organic reason for her symptoms, i.e., normal brain MRIs, normal lumbar puncture, she had a number of neurologists who examined her very carefully, such as Dr. Urrutia and Dr. Yan-Go. Id. at 645. Petitioner did not have stigmata of a neurologic injury. Id. at 646. If she had a brain injury, she should have had spasticity, abnormal changes in muscle tone, abnormalities in reflexes, abnormalities in pupillary function, fixed language deficits, and fixed problems. Her EEG in late 2009 was normal. All her brain CT scans and brain MRIs were normal. Id. All her cardiologic examinations, including EKG and stress echo on December 21, 2009, were normal. Id. at 647. Petitioner appeared to pass out at the end of her stress echo, which Dr. Lancaster interprets as another manifestation of her conversion disorder since her test results were normal and they did not document any hypotension during the study. Id. at 648. The cardiologist Dr. Tanenbaum found that petitioner's heart had a regular rhythm, normal cardiac sounds except for a low-grade murmur, with a blood pressure of 110/70 supine and 102/70 sitting, both of which are normal. Id. at 649-50. There was no objective evidence of autonomic neuropathy. The stress echo results of normal heart rate and normal blood pressure mediate against a diagnosis of autonomic disorder involving cardiovascular function. Id. at 650. Dr. Lancaster explained Dr. Tanenbaum's notation of neurocardiogenic syncope as a generic term for someone who appeared to pass out. Id. at 651.

Dr. Lancaster said that during petitioner's first three hospitalizations in September 2009 (twice at Inova Loudon and once at Inova Fairfax), when she complained of syncope or near syncope, the hospitals checked her orthostatic status and found nothing clinically significant. Dr. Lancaster did not see any objective testing revealing petitioner had significantly abnormal blood pressure or heart rate during the four months post-vaccination on August 23, 2009. Id. He explains petitioner's complaints as a result of conversion disorder. Id. at 652.

Dr. Jonathan Bresner, a neurologist at Inova Fairfax during petitioner's hospitalization from September 26-29, 2009, noted petitioner's tremulousness and abnormal body movements. Id. at 652-53. Dr. Lancaster commented that Dr. Bresner thought they were peculiar and difficult to characterize neurologically and regarded petitioner's neurologic exam as normal. Id. at 653. Dr. Bresner noted that he originally asked to see petitioner to check whether she had

GBS and decided she did not. He considered she might have a psychogenic etiology for her symptoms. Dr. Bresner thought petitioner might benefit from psychological counseling or psychiatric evaluation. Id. The discharge diagnosis was malaise and fatigue, abnormal involuntary movement not otherwise specified and lack of coordination, other malaise and fatigue, conversion disorder, obstructive sleep apnea, and stuttering. Id. at 653-54.

The discharge summary notes:

> At the time of presentation, the patient was noted to have very odd neurological symptoms with difficulty walking and tremors during evaluation of strength. However, patient's reflexes were normal and no other focal findings could be identified.

Id. at 654. The notes continue that Dr. Bresner suspected petitioner's neurological symptoms were not consistent with GBS and there could be a psychogenic component to her symptoms. Id. at 654-55. The records also note that petitioner was unable to hold a cup of water in her hand without spilling the water, but she was able to put makeup on fully in the morning during the hospitalization, including eyeliner, without complication until the nursing staff confronted her, after which she could no longer do this task. Id. at 655. The doctors did repeat testing of ANA and a phospholipid antibody panel. The doctor spoke to petitioner's personal care physician Dr. Kelly Rodriguez who did not see clear evidence of an obvious organic etiology, seemed concerned about a psychogenic etiology, and agreed with petitioner's having a psychiatric evaluation. Id.

Dr. Lancaster remarked that it is clear that many different physicians came to the same conclusion of a diagnosis of conversion disorder. Id. at 655-67.

Dr. Lancaster then turned to petitioner's visit to Johns Hopkins Hospital on October 2, 2009. Id. at 658. Petitioner was referred for an evaluation of GBS. Id. at 659. Petitioner reported bilateral lower leg weakness that progressed to the upper legs, increasing difficulty speaking and walking, and shortness of breath. Id. Petitioner had normal strength, sensation, and reflexes. Id. at 660. These were careful examinations to determine whether she had peripheral neuropathy and there was no evidence of it. Moreover, they did a lumbar puncture to rule out elevated protein. There was no GBS. Id.

Dr. Lancaster with reference to Dr. Steinman's thesis that there is a purely autonomic GBS said:

> I don't believe that anyone could make the diagnosis of a purely autonomic GBS. I don't believe that's an accepted medical illness, so I don't think we should rely on GBS or anything like that to explain her symptoms. [T]here's nothing about this case that makes any sense for GBS.

Id. at 661.

Dr. Lancaster said that Dr. Urrutia, the neurologist at Johns Hopkins, is a skilled neurologist who observed petitioner when she had her most active motor symptoms. Id. at 662. Dr. Urrutia did not find any objective deficits on petitioner's neurological examination. He observed that her symptoms clearly had a strong psychogenic component and he seemed to be working toward a diagnosis of conversion disorder. Dr. Urrutia wrote this note on October 3, 2009. Id. At a later time, Dr. Urrutia came by and noticed that petitioner felt better, having been given Ativan. Id. at 662-63. Petitioner had no focal deficits and was able to get off the bed walking normally backward or sideways, but not forward. Id. at 663. A physical therapist mentioned dystonia. Petitioner continued to stutter on examination but talked normally if she whispered. When she walked forward, she buckled and seemed jerky, but did not fall or hurt herself. She managed to turn around and sit on the bed. Dr. Urrutia's assessment was that her symptoms did not fit a physiologic paradigm and he thought she might be having a reaction to anxiety. Id.

Dr. Lancaster found it very interesting to see a neurologist who got to see things that were probably very similar to what Dr. Lancaster sees in his practice and what Dr. Lancaster observed in petitioner's videos reach the same conclusion he has. Id. at 663-64. Dr. Lancaster thinks significant Dr. Urrutia's description of petitioner's wild lurches in astasia-abasia, but she catches herself without injury. Id. at 664. Patients with organic disorders of gait unsteadiness fall down a lot and get hurt a lot. Id. Dr. Lancaster noted that even though the physical therapist at Johns Hopkins opined petitioner might have dystonia, none of the physicians at Johns Hopkins diagnosed petitioner with dystonia. Id. at 665. Dr. Lancaster agrees from his viewing of petitioner's videos that she did not have dystonia, and the treating neurologists' opinions are a helpful supporting factor. Id.

Dr. Lancaster reviewed petitioner's personal care physician Dr. Ho's records and noted that Dr. Ho did not independently assess and diagnose petitioner with dystonia. Id. at 669. Dr. Ho listed dystonia in his records because petitioner gave him a history that a physical therapist had told her this at Johns Hopkins. Dr. Ho recommended further workups which, if they proved unavailing, he would strongly consider a conversion disorder, delusional disorder, or other psychogenic etiology. Id. Even if petitioner were diagnosed with dystonia, Dr. Lancaster said that would not mean she had dysautonomia because dystonia and dysautonomia are very different. Id. at 670-71. Dr. Ho prescribed Valium, a benzodiazepine, used for anxiety among other reasons for petitioner. Id. at 671. Johns Hopkins had discharged petitioner on Clonazepam, which is another benzodiazepine, 0.25mg. Petitioner said she took the first dose and had convulsions afterward. She tried one-half dose the day before the visit to Dr. Ho, which was better, but she noted that when she took one of her then-husband's 5mg doses of Valium, she was "perfectly normal" for about 10 hours. Id. Petitioner said she tried some Ativan, which knocked her out. Id. She told Dr. Ho she planned to see a dystonia specialist at the Mayo Clinic in November 2009. Id. at 671-72.

Dr. Lancaster said he does not know how to provide a physiological mechanism to explain how taking a benzodiazepine would rapidly produce convulsions. Tr. at 672. He stated doctors use benzodiazepine to treat seizures. Benzodiazepines produce increased inhibition in

the brain. Dr. Lancaster strongly suspected that petitioner's convulsions were the same kind of event all her videos portrayed. He noted that benzodiazepines can be used to treat anxiety and that someone with conversion disorder would transiently feel somewhat better after taking a benzodiazepine or, in petitioner's case, with any other drug where there was a suggestion it might work. Id.

Dr. Lancaster noted petitioner's visit to Dr. Randolph Stephenson, a neurologist, on December 7, 2009, during which Dr. Stephenson gave petitioner a thorough examination which was completely normal. Id. at 673. However, when he asked petitioner to point or write something, she would start shaking quite vigorously as would her hands. Id. at 673-74. Petitioner's tremor was in several planes of direction and highly distractible. Id. at 674. It also had a highly suggestible component to it. Dr. Stephenson could easily bring her out of a tremor by having petitioner talk about it. Dr. Stephenson also recognized petitioner's gait was very clearly astasia-abasia. Petitioner would often appear off-balance, but she actually had better balance than the average person given her body position when she was walking. Dr. Lancaster's opinion was that Dr. Stephenson's record shows that petitioner's tremors were suggestible and distractible, meaning they were psychogenic. Id. A patient with psychogenic tremor who is suddenly distracted tends not to maintain the tremor. Id. at 675. Dr. Lancaster also noted that the fact petitioner's tremor was in multiple different directions and planes is very unusual for an organic tremor. Organic tremors tend to be very consistent. Id.

Dr. Lancaster commented that Dr. Stephenson's description of petitioner's gait was excellent, entirely consistent with astasia-abasia, and exactly what Dr. Lancaster saw in petitioner's videos. Id. at 676. He said that Dr. Stephenson's conclusion was that there was a very clear functional component to the majority of petitioner's physical examination. Id. at 676-77. "Functional component" means psychological component. Id. at 677.

Respondent's counsel then played Exhibit GGG, a video of Inside Edition, which aired on October 16, 2009. Id. at 678. Dr. Lancaster noted that petitioner's gait did not represent true classic dystonia. From the video, Dr. Lancaster said petitioner does not appear to have any fixed dystonic postures in any of her limbs. She has rather wild-flowing movements. She had deep bending of her knees and balancing. He remarked how much sheer energy it would take to do her gait. Id. He does not believe it is dystonic. Id. at 679. He would call it psychogenic dystonic. Dr. Lancaster also said that petitioner's gait and movements are not consistent with autonomic failure. He stated someone with autonomic failure has nothing directly wrong with the motor control of his or her legs and arms. The problem is with maintaining blood pressure. The person might get a little lightheaded, need to sit down and rest, but would not have anything like what petitioner manifested in the video from autonomic failure. Dr. Lancaster was impressed with petitioner's good autonomic cardiovascular control to behave as she did in the video. She did not report she was getting dizzy or needed to sit down or that she passed out. She did not have autonomic failure. Id.

Dr. Lancaster also did not think the symptoms petitioner depicted in the video supported a diagnosis of myasthenia gravis. Id. He called attention to her harsh speech and how much her face moved when she was trying to talk, while she was obviously moving very well considering

how much she was exerting herself. Id. at 680. Dr. Lancaster said myasthenia gravis does not get better with exercise. Myasthenia gravis gets worse with exercise. Id.

Although petitioner testified about weakness, Dr. Lancaster said he did not see any convincing evidence of weakness in the video and the other videos he saw. She seemed to be doing remarkably well in many aspects of strength. Being able to run on a narrow base with her knees deeply bent while jerking her arms takes a lot of strength. He does not believe she had muscle weakness in the video, and her physical examinations showed she had good strength in many muscle groups. Id. Dr. Lancaster stated the video is consistent with conversion disorder but not organic disease. Id. at 681.

Dr. Lancaster agreed that petitioner had rhabdomyolysis. She recovered from it in about two weeks. Her rhabdomyolysis was relatively mild. Id. Dr. Lancaster believes the cause of petitioner's rhabdomyolysis was a viral infection. Id. at 682. She was coughing up green phlegm on multiple occasions, had a sore throat day after day and subjective fevers. Exercise can be a very strong contributing factor to rhabdomyolysis. Petitioner was training for a race and she would probably have been fine if she did not have a cold. Muscle enzymes tend to rise with any significant strenuous exertion and superimposing a cold can lead to rhabdomyolysis. Id. Dr. Lancaster explained that rhabdomyolysis is a breakdown in muscle fibers from a muscle injury. Id. at 583. Typical causes are infection, overexertion or overuse, excessive heat, and severe metabolic derangements. Rhabdomyolysis reflects transient damage to the muscle. Id. The number one cause for rhabdomyolysis is infection and overexertion. Id. at 684.

Dr. Lancaster said it would be extremely unlikely for an autonomic nervous system problem to cause rhabdomyolysis. He stated the autonomic nervous system does not directly control skeletal muscle and could not make skeletal muscle break down. He remarked that nerve injuries of any sort tend not to cause rhabdomyolysis because the muscle is fine. Patients with GBS generally do not have a significant elevation in their creatine kinase (CK). Id. Creatine kinase is a muscle enzyme. Id. at 685. It is a useful marker for muscle injury. Too much protein in the blood from a damaged muscle could damage the kidneys. Id.

Dr. Lancaster testified that petitioner's rhabdomyolysis was a self-limited event and mild. Id. at 686. Petitioner's other symptoms have nothing to do with rhabdomyolysis. Id.

Dr. Lancaster then spoke about the Hallett article on psychogenic movement disorders (Ex. ZZ). Id. at 687. The first clue is movements may be inconsistent over time. This is something that both Dr. Stephenson and Dr. Lancaster noticed, the former during his physical examination and the latter while watching petitioner's videos. Tremors might come and go and be variable in frequency. The movements may sometimes be difficult to classify and may be mixtures of disorders such as myoclonus (rapid muscle jerks), chorea (dance-like movements) and dystonia. The movements might be so unusual as to be classified as bizarre. Movements might disappear with distraction or be precipitated by suggestion. Other features of psychogenic movement disorders are give-away weakness, which means inconsistent effort when a doctor is testing a patient's strength. Id. A patient who does not have an organic disease will often have a brief strong effort and then the limb will give way suddenly. Id. at 688.

Hallett also mentions psychogenic gait which is characterized by unusual patterns of stance and gait often inconsistent, often dramatic with lurching, but only rarely results in falls and then the patient does not hurt himself or herself. Sudden knee buckling without falling is a common pattern. Dr. Lancaster thinks the Hallett article is a good description of what he has seen in this case, i.e., a great variability in the types of tremors, the characteristics of tremors, the types of speech problems, the types of movement problems in general, and the gait where petitioner almost falls and does not hurt herself. This is all an excellent fit. Id.

Respondent's counsel then played the next video, Trial Exhibit 58-1, which is dated October 17, 2009, and depicts petitioner preparing for a race, her dysarthria is getting better, she walks backward to start the race, and starts the race jerking a little but soon her running turns into a normal run and she speaks normally while running an 8K race. Tr. at 689. Dr. Lancaster says that petitioner's presentation was inconsistent with autonomic dysfunction because there was no indication that she had trouble maintaining her blood pressure as she was running, she was talking quite clearly, and she did not report feeling lightheaded, dizzy, or weak. Dr. Lancaster stated that petitioner's presentation in this video is also inconsistent with dystonia. Id. Petitioner has a psychogenic movement disorder with a lot of lurching and apparent instability, but not real instability, and then she gets better. Id. at 689-90. Dr. Lancaster found novel the idea that vocal dystonia would improve when petitioner was running. Id. at 690. He is not aware that anyone has ever reported that for dystonia. Petitioner's presentation in the video is inconsistent with myasthenia gravis. Someone with myasthenia gravis would rapidly fatigue and need to stop running as he or she attempted to run. His or her speech would not improve while running. He or she would be short of breath and start gasping. Id. Dr. Lancaster did not see any weakness in this video. Id. at 691. Dr. Lancaster said that petitioner's presentation in the video was inconsistent with petitioner's having any organic neurologic condition. Id. Conversion disorder is a diagnosis doctors use when the symptoms have a psychiatric rather than a neurologic basis. Id. at 692.

Dr. Lancaster testified it would be extremely unlikely for someone with active myasthenia to run an 8K race. Id. at 693. He said that petitioner's presentation of symptoms in this video is inconsistent with an autoimmune etiology because autoimmune damage to the nervous system does not come and go this quickly. Id. at 694. Autoimmune diseases do not suddenly turn on and turn off.

Respondent's counsel then played the next video, Trial Exhibit 58-3, which is an interview after the 8K race on October 17, 2009. Id. Dr. Lancaster describes the video as showing petitioner sitting at the end of the race, describing how when she stops running, her abnormal movements return. It does not sound like cardiovascular collapse. Petitioner's breathing appears very steady and effortless. Her face has a strong expression. She does not have any evidence of ptosis, i.e., drooping of the eyelids. She appears to move her tongue normally when she talks. Dr. Lancaster said he never sees a focal paralysis of the tongue in myasthenia gravis. He could not think of any organic etiology for focal tongue paralysis. It is another symptom of conversion disorder. Id.

Respondent's counsel then played the next video, Trial Exhibit 58-4, also dated October 17, 2009, which shows petitioner walking backward slowly into a house while manifesting abnormal speech. Id. at 695-96. Dr. Lancaster stated petitioner walks backward as she did before, but before when she walked backward, she did that normally and effortlessly. Id. at 696. In this video, she walks backward stomping each time she takes a step backward. She lifts each leg high and stomps it down while leaning forward as she walks backward. Dr. Lancaster said it takes more strength and balance to walk backward upstairs than it does to walk forward or even forward up a staircase. This increases the amount of work and balance petitioner did by a couple of factors, arguing more against her having dystonia. Id. Dr. Lancaster said this video does not have anything to justify a diagnosis of either dysautonomia or myasthenia gravis. Id. at 697. Instead, it supports the diagnosis of conversion disorder. Id.

Respondent's counsel then played the next three videos, Trial Exhibits 58-5, 58-7, and 58-8, all dated October 17, 2009. Id. at 697-98. Trial Exhibit 58-5 depicts petitioner drinking water and eating food very quickly at a table, swallowing very well. Then she immediately drops her head and is assisted to a couch where she lies on her side and continues to eat while having "seizures." Then she starts having a lollipop. She says it was hard to breathe and then starts crying. Trial Exhibit 58-7 depicts petitioner lying on a couch, eating strawberries. Her then-husband holds her head down so she can continue to eat strawberries. Trial Exhibit 58-8 depicts petitioner's then-husband holding her forehead and chin while feeding her blueberries. Petitioner finished the whole bowl and has a "seizure" after moving her tongue. Her speech is slow and slurred.

Dr. Lancaster said that the tremors appearing in the videos are not dystonic because the pattern of complex shaking of multiple areas of petitioner's body progressing over time is inconsistent with dystonia. Tr. at 698. He stated dystonia tends to be relatively localized to certain muscles or groups of muscles. Id. at 698-99. He said that petitioner's convulsive-like event is not what dystonia looks like and is a psychogenic event. Id. at 699. An epileptic seizure causes patients to lose consciousness. Id. When petitioner was shaking in all four limbs but still wide awake, manipulating things with her arms, and responding to what other people were doing or saying to her, that is inconsistent with an epileptic seizure. Id. at 700. Where in petitioner's brain would this seizure have to be? It would have to be in the right hemisphere to make the left side of her body and head jerk. Id.

In addition, 90 percent of the time, people who have epileptic seizures open their eyes in the direction away from the hemisphere driving the seizure. Id. at 700-01. Only a small subset of people having an epileptic seizure have their eyes closed. Id. at 701. But petitioner at many points closed her eyes while she was shaking. Her limbs were shaking while she used her left hand to feed herself strawberries. Dr. Lancaster said this makes no sense. The videos show petitioner eating during a seizure that was affecting her neck and head muscles so that her head was jerking. Anyone who tried to eat during a seizure would most likely inhale the food into his or her lungs and would aspirate and choke. But the videos show how well petitioner ate multiple times. This is very consistent with a psychogenic seizure. Id.

Another very important factor is petitioner's idea that protruding her tongue caused her to seize. Id. Dr. Lancaster said this makes no sense because protruding the tongue does not cause epileptic seizures. Id. at 702. However, anything that the patient thinks can trigger a seizure can trigger a psychogenic seizure. Dr. Lancaster thought it interesting how well petitioner could stick out her tongue. When petitioner talked, her tongue often appeared paralyzed, but at other times, petitioner sticks out her tongue.

Dr. Lancaster noted petitioner was breathing very well. He said petitioner's swallowing and breathing functions were excellent. This is inconsistent with myasthenia gravis. Myasthenia gravis patients have trouble swallowing and if they attempted drinking a whole glass of water, they would choke. Myasthenia patients do not have shaking spells like this either. Similarly, autonomic dysfunction is not the cause of her symptoms. In autonomic dysfunction, the voluntary parts of swallowing are basically fine. Id.

The videos do not show a blood pressure drop or syncope even when petitioner is lying flat on her back. Id. at 703. Yet she is having all these complex movements. If she were passing out, she would not be feeding herself strawberries. Nothing about the video depicts myasthenia gravis or autonomic failure. If you showed 100 neurologists this video, a high percentage would say petitioner is having a non-epileptic seizure and no one would diagnose myasthenia gravis, autonomic failure, or dystonia. Id.

Dr. Lancaster said the videos show petitioner had really excellent use of her chewing muscles. Id. at 704. She very rapidly and strongly chewed her food. She swallowed both water and solid food without difficulty. Her jaw muscles were not weak. Id. Petitioner's presentation in this video is inconsistent with any known organic neurological condition. Id. at 705.

Respondent's counsel then played the next video, Trial Exhibit 58-12, dated October 18, 2009, which shows an interview continuing. Id. Petitioner's speech flows more than before. She does not show physical impediment and uses her hands while talking. Then she slows down and starts singing "Mary had a Little Lamb" but then transitions to a mini seizure that does not last long. Petitioner goes out of head bopping and hand curling to talk to the interviewer again. Then she stands to demonstrate that she does not feel lightheaded standing, but only when she starts trying to move.

Dr. Lancaster says that petitioner is discussing how moving her tongue triggers seizure-like events. She has a vocal problem which continues to fluctuate under different circumstances. Here, she is speaking in a very strong, loud voice with good movements of her face. But she often has a harsh tone in her voice that seems to go away instantly when she touches her jaw. Then when she sings, the movement returns with shaking of the head. Id. Her arm movements are good and normal. Id. at 706. Petitioner's presentation in this video is not consistent with dysautonomia, dystonia, and myasthenia gravis. She has excellent strength in her face as she is moving, her eyelids do not droop, and she does not have respiratory distress. Someone with myasthenia gravis who tries to sing will get worse because you have to move more air and use your vocal apparatus more to sing than to speak. Id. Patients with myasthenia gravis give up

singing (referring to Ex. WW, at 3). Id. at 708. Dr. Lancaster said that petitioner's presentation in the video is inconsistent with any known organic neurologic condition. Id. at 709.

Respondent's counsel then played the next two videos, Exhibit AAA and Trial Exhibit 58-20. Id. at 710. Exhibit AAA is the 20/20 broadcast of clips of petitioner stumbling and struggling to walk, and clips of an interview of petitioner and her then-husband in which petitioner speaks in a British accent. Then there is an analysis of whether she is faking it or not. There are clips of an interview with Dr. Buttar defending his treatments and allegations he is a fraud. Experts state petitioner does not have dystonia and that whatever she has is not caused by flu vaccine. Clips comment on her Internet fame and petitioner comments that there is no way she is faking it because she would have to be an amazing actress to do this. Other experts in the clips believe it is psychogenic. Trial Exhibit 58-20, dated October 18, 2009, shows petitioner and her then-husband sitting on a couch using a laptop together. Petitioner has the laptop on her lap with earphones in. Petitioner has slurred speech. She is reading e-mails from people contacting her regarding her condition including doctors and sympathizers. Petitioner says it is hard to read, but she types on her laptop well.

Dr. Lancaster notes that petitioner's speech pattern changed again in that it was starting and stopping with a staccato quality. He noticed petitioner seemed to be using her computer without difficulty. She and her then-husband seem to be learning about Dr. Buttar's clinic. Id. Petitioner mentions that some patient received detoxification treatment, saying "her got detox." Id. at 711. To Dr. Lancaster, this indicates not only trouble pronouncing, but also an abnormal word choice. Petitioner complained about her eyes jerking or it was difficult for her to read. That would require an explanation for what happened to her ocular pathways, which Dr. Lancaster said he has yet to hear. None of petitioner's medical records, including neurologists' notes and other doctors' notes, say there is anything objectively wrong with petitioner's visual system either in her eye movements or in terms of her visual acuity. To give petitioner an organic etiology for her symptoms would require giving her "a pile of different diseases that are located in many different areas of the nervous system." Id.

Dr. Lancaster stated that petitioner's symptoms have been fluctuating and changing the entire time, depending on the situation and what she is doing. Id. at 712. Sometimes she has a lot of tremors in her arms. Other times, her arms are fine. Sometimes, she has a neck tremor. Other times she does not have a neck tremor. Sometimes her speech problem is foreign accent syndrome where she speaks clearly but with a British accent. Other times, her speech is harsh and strained. Still other times, her speech is slow and halting. Throughout these videos, petitioner's symptoms are constantly different.

The autonomic nervous system does not process the muscles that allow one's eyes to move from side to side. Those muscles are under voluntary control. Similarly, the perception of light is not considered part of the neurologic problem. The only part of the autonomic nervous system relating to vision is the generation of sufficient tears. If that fails, someone would get terribly dry eyes or dilated or constricted pupils. Id. Petitioner has been examined multiple times with no evidence of any defect. Id. at 713.

Dr. Lancaster said that dystonia, dysautonomia, and myasthenia gravis are not relevant to her gait problems, speech problems, perception problems, abnormal feeling of hot and cold (sensory symptoms), language problems, and problems thinking what word she is going to say. Id.

Respondent's counsel then asked about petitioner's treatment at Dr. Buttar's clinic. Dr. Lancaster described chelation as using a chelator, a substance for binding heavy metals, on a patient who is actually exposed to heavy metals. Id. at 714. Chelators are chemicals which in high enough doses to work are quite dangerous and potentially toxic. Dr. Lancaster called them serious substances. He does not know what dose Dr. Buttar used on petitioner. Id.

Dr. Buttar had two chelators. Id. at 716. Dr. Lancaster said, "the idea that you could get enough of these things to be physiologically effective by putting the drops on your arms is ridiculous." Id. The little drops would not make any difference at all.

In addition, Dr. Buttar put petitioner in a hyperbaric chamber with pressurized air, but not hyperbaric oxygen. Id. Petitioner received several IVs, the exact contents of which are unclear. Id. at 717. Most importantly, Dr. Lancaster said, Dr. Buttar repeatedly suggested to petitioner that she would respond to different treatments and recover. He said the DMPS drops (another chelator) have no validity or use. Id. The lollipop (Vitapop) that petitioner was sucking in a video with vitamin B12 would have no effect on a person who was not vitamin B12 deficient. Id. at 718. The idea that someone could suck on a vitamin B12 lollipop and have an instantaneous effect is the placebo effect. Id. Dr. Lancaster said petitioner did not have mercury poisoning. Id. at 719. She was never exposed to any sufficient source of mercury to cause her symptoms unless she ate one or two thermometers which she never reported. The amount of mercury and the type of mercury in vaccines is completely harmless. Dr. Buttar said he was chelating petitioner because of ethyl mercury in flu vaccine. Id.

Dr. Lancaster remembered one treatment at Dr. Buttar's clinic purportedly to remove blood from one of petitioner's arms and return it to the other arm after passing it through a machine. Id. at 720. Dr. Buttar was going to blast the blood with ultraviolet light to kill whatever infection petitioner might have. Id. Dr. Lancaster said he had no idea what this machine actually did. Id. at 720-21. It did not correspond to any accepted medical treatment for infection of which he is aware. Id. at 721. It sounded hazardous to Dr. Lancaster in that it could introduce a blood clot by destroying petitioner's blood cells. It could create a sepsis response. He could not imagine it helping with any infection. Dr. Lancaster said that sitting in a pressurized air cubicle would be very unlikely helpful and he agreed with Dr. Steinman that the accepted medical use of hyperbaric chambers is primarily for treating diving injuries. It also can potentially accelerate wound healing. Id. He has never used it for an autoimmune disease. Id. at 722. Dr. Lancaster believes Dr. Buttar's diagnosis is completely incorrect and he would not have given any of those treatments for whatever diagnosis petitioner had. Id.

Respondent's counsel then played the next video, Trial Exhibit 58-35, dated October 20, 2009. Id. Dr. Lancaster described the video as depicting petitioner undergoing treatment in the hyperbaric oxygen chamber. Id. at 723. Stan Kurtz says this is the first time he has heard

petitioner speak in a normal tone of voice. Petitioner's symptoms have gone away. She is eating normally, with excellent use of her hand to control the fork and no difficulty with coordination. She can now stick out her tongue and wag it around without it triggering symptoms or abnormal movements. Petitioner appears not to have respiratory distress or weakness of breathing. She swallows her food perfectly. Id. She does not vomit. Id. at 724. Before then, petitioner was speaking abnormally. Dr. Lancaster said that hyperbaric treatment is not effective for dystonia, autoimmune encephalitis, autonomic disorders, myasthenia gravis, or any other known autoimmune disease. Id. Dr. Lancaster said petitioner's response to the hyperbaric treatment in this video is not consistent with an autoimmune condition. Id. at 724-25.

Dr. Lancaster explained that autoimmune conditions involve either antibodies that bind to specific targets and nerves, i.e., neuromuscular junction in myasthenia gravis, autonomic ganglia in autoimmune autonomic neuropathy. Id. at 725. They use complement and other mechanisms to cause lasting damage at those locations. If someone could use a magic wand to dispel instantly antibodies completely from someone with an autoimmune disease, such as myasthenia gravis, it would take days to weeks for the neuromuscular junctions to be repaired from the damage the antibodies did. Someone with autoimmune autonomic neuropathy would take days, weeks, or months to recover from an elimination of all those antibodies. Patients with autoimmune encephalitis have antibodies affecting the brain causing long-lasting changes in ion channels. That would take days, weeks, or months to become totally better as well. Id. The idea that an antibody-mediated disease would instantly resolve from any mechanism makes no sense. Id. at 726.

Dr. Lancaster said that if we are talking about cytotoxic T-cells damaging other cells, there is even more lasting damage which will not get better instantly even if one could make the T-cells immediately disappear. Improvement would take days, weeks, or months of recovery. Dr. Lancaster stated petitioner's very rapid recovery in the video between profound disability and being totally fine mediates very strongly against her having any autoimmune neurologic disorder in his experience. He thinks the reason petitioner improved with chamber therapy was the placebo effect because of the very strong suggestion that she would improve. One can hear in the video that petitioner was being told how these things would work over and over. Id. In a psychogenic illness, suggestion is powerful. Id. at 727. The following colloquy took place:

> THE COURT: I take it the basis of your testimony that she doesn't have what she alleges she has, dysautonomia, dystonia, myasthenia gravis, is not only that the symptoms that you're familiar with from the medical records do not match those illnesses, but that she also recovered instantaneously with this fake treatment?
>
> THE WITNESS: That is exactly my point. And that's not consistent with any of these diseases. There is nothing that I can conceive of that would make those diseases instantly get better.

Id.

Respondent's counsel then played the next video, Trial Exhibit 58-46. Id. at 728. This video deals with petitioner's assertion that she had cold spots in her head. Dr. Lancaster said that myasthenia gravis would not cause cold spots or abnormal cold sensation in her head because myasthenia gravis is a motor problem not a sensory problem. Id. He would not associate cold spots with dystonia or any autonomic symptoms either. Id. at 728-29. Dr. Lancaster thinks petitioner is reporting sensory symptoms for which an organic basis has not been established. Id. at 729. Petitioner was trying to make sense of her symptoms through the idea Dr. Buttar gave her of mercury coming out of her body. Id. at 730. Dr. Lancaster's best explanation is the power of suggestion. Dr. Lancaster noted that the video showed petitioner awake, alert, speaking normally and clearly, and using her face normally. Id.

Respondent's counsel then played the next video, Trial Exhibit 58-50. Id. at 731. This shows petitioner walking forward, backward, and sideways, telling her then-husband she feels good and talking on a cellphone coherently and loudly. Dr. Lancaster states petitioner's speech function, facial movements, limb movements, gait, and balance have completely resolved with her treatment at Dr. Buttar's clinic, which is very rapid. Id. Dr. Lancaster said that various IV fluids and glutathione, which is an antioxidant, would be highly unlikely to have had any physiologic effect on the alleged diseases. Id. at 732. This is another factor favoring psychogenic etiology. Id.

Respondent's counsel then played three videos, Trial Exhibits 58-59, 58-60, and 58-61, all dated October 21, 2009. Id. at 732-33. Petitioner is lying on a couch, feeling unwell, struggling to speak. Dr. Buttar arrives at the hotel with DMPS transdermal drops which he applies to petitioner's arms. Petitioner lying on the couch speaks and says her tongue is coming back and her speech starts to improve. They tell her it was the mercury. She says her lungs are burning and it will cause a seizure. She indicates the side of her mouth is burning and her speech becomes slurred again. Dr. Buttar is getting ready to give her more drops, saying petitioner is so toxic that it is phasing fast. Petitioner says it is moving to the back of her head and her speech is comprehensible again. Dr. Buttar puts more drops on her arms and she rubs the drops in herself. In the middle of petitioner's rubbing, her speech worsens suddenly and is a raspy moan. Then within a minute, her speech becomes normal again. She talks about the path the burning sensation is traveling and how it differs with each dose. Dr. Buttar offers to have petitioner and her then-husband stay at his home if the drops do not help.

Dr. Lancaster described these DMPS drops as chelator drops. Id. at 733. He notes how Dr. Buttar repeatedly suggested that they would work. Dr. Buttar also told her that she had mercury toxicity and that mercury was being removed from her body and was moving through her body. Id. Dr. Lancaster said several things were not plausible. Id. at 734. Someone with mercury toxicity cannot feel it moving throughout his or her body. Mercury would not affect how someone feels in an MRI. Mercury would not heat up and, if it did, it would be so evenly distributed throughout someone's body that someone would not notice it. The idea that a chelator would get into someone's body and move mercury around which would somehow cure a patient of any neurologic disease is completely implausible, said Dr. Lancaster. Dr. Buttar told petitioner all sorts of things happening to her body and she developed symptoms corresponding

to the story he told her about mercury coming out of her body. He told her the drops would work and they did through the placebo effect. The videos show petitioner moving her arms, at first slowly and clumsily, and then just fine after the application of more drops. Id.

Dr. Lancaster said that petitioner's speech problems, always a little different, manifested as a very slow voice that would taper off and slow down until she stopped talking. Id. at 734-35. At times, petitioner had a harsh croaking sound and then she would rapidly return to her normal voice at different points of the treatment. None of this is biologically plausible. Id.

Dr. Lancaster said that autonomic failure would not cause that kind of speech problem and would not respond to the placebo effect. Id. at 736. Myasthenia gravis does not cause that sort of speech problem and would not respond on and off very quickly like that to anything. But conversion disorder frequently would respond to suggestions and placebo effects. Dr. Lancaster said that what petitioner is manifesting does not make medical sense, in other words, is not logical as an organic illness. Id.

Respondent's counsel then played the next video, Trial Exhibit 58-62, dated October 22, 2009. Id. at 739. Petitioner is eating on a couch, using utensils and speaking normally. Petitioner says she got tired and had a good night because she could fall asleep. She felt great. Dr. Lancaster's observation was that petitioner appeared perfectly well. She was clearly feeling fine, sitting up, and eating. She did not have any problem chewing, swallowing, or digesting (no vomiting). Id. Petitioner mentions a seizure that moving her eyes or reading triggered. Id. at 740. Dr. Lancaster said that unusual triggers fit within the paradigm of psychogenic, non-epileptic events. He did not see any symptom in the video of dystonia or myasthenia gravis. Id.

Respondent's counsel then played the next video, Trial Exhibit 58-63, dated October 22, 2009. Id. at 741. It depicts petitioner in a car with her head bobbing that moving her tongue induced. Dr. Lancaster stated petitioner had some neck thrusting movements. He said this would be extremely atypical for dystonia and even more atypical for dystonia is petitioner's claim she could trigger the neck thrusting movements by moving her tongue. On top of that, petitioner claims her tongue is numb, which autonomic failure, myasthenia gravis, or dystonia would not cause. There is no organic explanation for this. Id. The idea that someone could move his or her tongue and then have involuntary head thrusting does not correspond to any neurologic disease Dr. Lancaster knows. Id. at 742.

Respondent's counsel then played the next video, Trial Exhibit 58-65, dated October 22, 2009. Id. Petitioner has finished eating while receiving an IV. She does not have the electrical device on her neck. She first speaks normally, but then pauses, is quiet, and starts whispering. Then she seems to have a seizure. Her head bobs and her speech is slurred. In less than one minute, she can speak normally. Then petitioner switches back and forth within seconds between normal and slurred speech. Id. Dr. Lancaster stated the video shows petitioner's abnormal head thrusting and changes in her voice fluctuating very rapidly, turning on and off. She does touch her chin, which is her understanding of a sensory trick working for dystonia. Id. at 743. Dr. Lancaster said that cervical dystonia does not look like that. Cervical dystonia involves a forced turn and/or tilt of the head to one side. It tends to be relatively fixed and is

very uncomfortable. Torticollis is a form of cervical dystonia. Petitioner's repeated head thrusting is not cervical dystonia. She read something about sensory tricks and that they are supposed to work. Id. This is one of the many factors that support Dr. Lancaster's diagnosis of conversion disorder. Id. at 744. Petitioner was told she had arsenic poisoning and uranium poisoning which he finds implausible. Id. Dr. Lancaster testified that the video does not show any evidence of actual autonomic disease. Id. at 744-45. Nothing about the autonomic nervous system would explain what petitioner manifests in this video. Tr. at 745.

Dr. Lancaster said that a patient with myasthenia gravis could have a head drop, which looks very different than petitioner's thrusting of her head. Head drop is gradual so that the chin rests on the chest or near it and it takes more effort to bring the head up. Id.

Respondent's counsel then played the next video, Trial Exhibit 58-66. Id. at 746. Petitioner is sitting in her treatment chair, talking coherently, and shaking a bottle of transdermal DMPS drops, saying "This stuff saved my life." She says she used to have 20-25 seizures daily and it stopped when a drop of transdermal DMPS was on her skin. Dr. Lancaster says this is additional confirmation that the treatment had a very powerful placebo effect on her symptoms. Id.

Respondent's counsel then played the next video, Exhibit DDD, dated October 29, 2009. Id. at 747. This video shows petitioner receiving IV treatment at Dr. Buttar's office, thanking people and wanting to let people know that she was getting better. She speaks normally. The video was going to a website where she posted status updates of her health. Dr. Lancaster comments petitioner reports feeling quite well, she is sitting upright, and she is talking and thinking clearly and logically. Id. at 748. She does not have any evidence of weakness of the muscles of her face. Her breathing is relaxed and at a normal rate. She appears well. There is no evidence that she has dysautonomia, dystonia, or myasthenia gravis. Id.

Respondent's counsel then played the next video, Exhibit JJJ, which is a Fox 5 news clip that aired on November 19, 2009, but the date Fox filmed it is unknown. Id. It details petitioner's progress in Dr. Buttar's office. Her voice changes whenever she talks about the time period when she believes her injury occurred, which Dr. Lancaster states is implausible for dystonia to appear instantly and result in a slowing of speech, much less that saying a particular word, such as the date something happened, would trigger dystonia. Id. at 749. He also said that myasthenia gravis would not suddenly come on and impair someone's speech as the person said a particular word. He said it was a ridiculous concept. Dr. Lancaster stated that the idea that autoimmune autonomic neuropathy would change as someone says a word affecting his or her speech is "ridiculous squared. It's so absurd I don't even know how to explain how illogical it is." Id. The reason for petitioner's sudden speech symptom to come on when she mentioned a date is psychogenic. She knew it was stressful to talk about. Id.

Dr. Lancaster stated that the idea that remembering something would trigger vocal dystonia is incorrect and not logical. Id. at 750. The idea that a memory would affect myasthenia gravis is illogical. The idea that a memory would affect an autonomic system disorder such as autoimmune autonomic neuropathy is completely implausible. These are

problems with how ganglia are working in the sympathetic and parasympathetic nervous systems. They do not care what one's memories are. It makes no medical sense. Id.

In recapping his analysis of all petitioner's videos, Dr. Lancaster said petitioner did not have epileptic seizures. Id. at 751. Her tremors were psychogenic, resembling seizures. Id. From October 17 through October 22, 2009, the videos did not show any evidence of petitioner having respiratory distress or breathing problems. Id. at 751-52. He did not see her lose consciousness. Id. at 752. She had episodes he would describe as non-epileptic seizures where her consciousness appeared to be altered, although she would partially respond during those events. Someone with an autoimmune disorder would not respond rapidly to treatments that Dr. Buttar administered. Id. Her symptoms fluctuated: seizure-like events, abnormal movements involving gait, apparent alterations in her level of consciousness, abnormal tremors in the limbs, and abnormal positive and negative sensations such as feeling cold spots moving around her body, or feelings of heat in the MRI. Id. at 752-53.

All of these cannot be localized to any one part of the nervous system. Id. at 753. She responded strongly to suggestion about what would happen. The overall nature of the events was consistent with psychogenic movement disorder, astasia-abasia, psychogenic seizure-like activities, and psychogenic tumors. There is only one logical conclusion, i.e., petitioner had conversion disorder fed by repeated suggestions about the sort of disease process she was thought to have, such as mercury poisoning, and fed by what thoughts she had about her responses to treatment which Dr. Buttar and other people gave her. Id.

Dr. Lancaster disagrees with petitioner's neurologist Dr. Cintron's assessment that petitioner had a vaccination-induced motor disorder because there is no diagnostic test to support that conclusion. Id. at 755. Dr. Lancaster believes from the videos that petitioner had a couple of dramatically different gait fluctuations. Id. at 757. Dr. Cintron would not have seen these inconsistencies because he did not view the videos. Id.

Although Dr. Cintron recommended plasmapheresis and IVIG treatment, Dr. Lancaster does not agree with those recommendations. Id. at 757-58. Dr. Cintron did not go forward with those recommendations. Id. at 758. Dr. Lancaster stated that Dr. Cintron did not mention the autonomic nervous system at all in his notes. He mentioned a motor disorder. Id. A motor disorder involves voluntary movement, very different from a disorder of the autonomic nervous system. Id. at 759.

When petitioner was at the Inova Loudoun Hospital ED on September 17, 2009, Dr. Dulai, a neurologist, did a physical examination and found petitioner did not have significant weakness. Id. at 761-62. This is inconsistent with myasthenia gravis. Id. at 762. Her eye movements and eyelids were basically intact. Id. Dr. Dulai's examination results are not consistent with dysautonomia. Id. at 763. Her vital signs were normal. Id.

Dr. Lancaster testified that from August 23, 2009, when she received flu vaccine, through the end of 2009, petitioner did not have autoimmune failure or myasthenia gravis. Id. at 764. In that period of time, she did not have dystonia unless it is described as psychogenic dystonia. Id. at 766. She had conversion disorder. Id.

On the fourth day of the hearing, Dr. Lancaster resumed his direct testimony. Id. at 773. Respondent asked Dr. Lancaster if an article marked as Exhibit 176 was relevant to petitioner. That is the Raj article on blood volume in POTS. The authors state that patients with POTS have chronic symptoms lasting at least six months, consisting of rapid palpitation, exercise intolerance, lightheadedness, extreme fatigue, and mental clouding. They have an increase in heart rate of at least 30 beats/min within 5 to 30 minutes of assuming an upright posture which should occur in the absence of orthostatic hypotension, i.e., a fall in blood pressure >20/10 mm Hg. The authors also state many patients with POTS have low blood volume. The authors theorize that abnormalities in the renin-angiotensin-aldosterone axis may play a role in the pathophysiology of POTS by contributing to hypovolemia, perhaps by impaired sodium retention. They trace perturbations in the renin-aldosterone system to partial sympathetic denervation involving the kidney, an explanation consistent with the partial dysautonomia hypothesis for some patients with POTS. The recommendation to increase salt in the diet and intake of water is commonly made for POTS patients.

Dr. Lancaster said the Raj article does not explain what happened with petitioner. Tr. at 774. The basic idea of the article is that patients with POTS have a mild degree of cardiovascular autonomic dysregulation for reasons not entirely known. It is a syndrome but not a disease. Id. He agrees with Dr. Steinman that giving a patient with POTS IV fluid for a long period of time lessens POTS symptoms. Id. at 775. Feeling better would only deal with lightheadedness and dizziness. It would not deal with foreign accent syndrome, complex movements, and numbness. In Dr. Buttar's office, petitioner did not really seem to have any symptoms consistent with POTS and she had numerous symptoms that POTS would not explain. Id.

Dr. Lancaster said that someone with POTS might feel a little tremulous or shaky when standing up and need to sit down. Id. at 776. But the violent tremors petitioner exhibited were not indicative of POTS. Psychogenic seizures are not related to POTS either. IV fluids would not help them physiologically. But someone could have a placebo effect through receiving IV fluids which could likely work with psychogenic seizures. Id.

Dr. Lancaster stated it was extremely unlikely that petitioner had POTS when she was receiving treatment at Dr. Buttar's clinic. POTS could not plausibly account for the symptoms appearing on the videos. Dr. Lancaster does not believe petitioner ever had POTS. Id. The numerous times when petitioner had various tests and maneuvers, she developed numerous symptoms without having characteristic blood pressure changes or pulse changes one might expect with POTS, particularly the pulse changes. Id. at 776-77.

Moving beyond 2009 into 2010, respondent's counsel showed a video, Exhibit BBB, which is a segment of Inside Edition, aired February 4, 2010, depicting an encounter between the Inside Edition reporter and petitioner in a parking lot in January 2010. Id. at 777. The show is an update following petitioner and showing clips of her walking normally and driving. The reporter walked up to her and she told him she had recovered but not completely because she was speaking with an Australian accent. Petitioner insisted that her illness was not psychogenic. At the end of the interview, as she approaches her parked car, she walks sideways.

Dr. Lancaster found significant the clip of petitioner walking very normally without any sign of a movement disorder as she comes out of a store before meeting the reporter. Id. at 778. She speaks loudly and very clearly with what appears to be an Australian accent. After she finishes talking to the reporter, she walks sideways to get to her parked car. She also reported cognitive issues, but she was driving at the time and did not show any signs that Dr. Lancaster could observe of cognitive issues. Dr. Lancaster stated these observations are consistent with the overall picture of a psychogenic etiology. Petitioner did not have any symptoms until she encountered the reporter, a stressful situation, and he asked her uncomfortable questions, resulting in the recurrence of her symptoms such as the foreign accent. Id. Nothing in this video would support a diagnosis of dystonia, dysautonomia, POTS, or myasthenia gravis. Id. at 779.

Even though petitioner testified that her foreign accent was due to her inability to pronounce words, Dr. Lancaster said she pronounced words very well. He thinks many people speaking with an Australian accent would sound exactly like petitioner did. Dr. Lancaster stated in his career, he has heard many patients with different types of dysarthria, but their dysarthria did not make them sound like that. "Dysarthria" means difficulty pronouncing words without any impairment in thought content or language content. The words do not come out clearly as if someone had a bunch of marbles in his or her mouth. Id. Someone with a nerve injury that paralyzed his or her tongue would have dysarthria. Id. at 780. Someone with a stroke that weakened facial and mouth muscles could have dysarthria. An injury to the cerebellum would affect coordination of motor movements can cause dysarthria. Id. Petitioner sounded as if she were either Australian or someone trying to speak as an Australian.

As time moved on from 2010, petitioner's symptoms changed. Id. at 781. In the early time period, petitioner's predominant symptoms were abnormal lurching gait. That faded away or was much less prominent. Her complaints of seizure-like episodes which were very prominent then faded away. Complaints of cognitive clouding were initially very prominent but became much less prominent in subsequent visits. Id.

Then, reports of gastrointestinal or autonomic problems came on. In the beginning, she had trouble eating. Id. But she would say she had trouble when she moved her tongue because it triggered seizure-like events or abnormal neck movements or other symptoms. Id. at 782. At times, she said her tongue was numb or paralyzed. These were troubles of voluntary eating. But her initial reports would not have indicated gastroparesis where someone eats and vomits subsequently. Those symptoms emerged later, i.e., inability to keep food down after swallowing it. Dr. Lancaster distinguished between everything post-vaccination in 2009 and what petitioner complained of in 2010. Id.

In 2009, as petitioner described in the videos, she reported that movement of her tongue triggered abnormal thrusting movements of her head or sometimes seizure-like spells. This is the reason she gave for having trouble eating. Later, she did not connect her symptoms to tongue movement, but to her not keeping food down. Id. at 783. Petitioner saw Dr. Patrick D. Lyden, a neurologist, on January 10, 2012 for an evaluation of dysautonomia and spells. Id. at 784. Petitioner told him she developed POTS after a flu vaccination in 2009. Id. at 784-85. Petitioner gave a history of muscle weakness, fatigue, exercise intolerance, tachycardia, and postprandial

hypotension. Id. at 785. Dr. Lancaster stated Dr. Lyden was unaware of petitioner's lurching gait and seizure-like spells because petitioner did not tell him. She told him she had exercise intolerance but did not tell him her profound movement disorder got much better with exercise. Id. She did not tell him of numbness or tongue movement triggering her symptoms. Id. at 786. She did not tell him about dystonia. Dr. Lancaster thinks petitioner's report to Dr. Lyden of a positive tilt-table test is an oversimplification of what happened during the test. Dr. Lyden's view of the case, therefore, is different than if he had seen the videos filed in the case. Id.

Dr. Lyden stated in his records that the case was very complicated. He thought petitioner might have severe dysautonomia and a possible diagnosis of migraine. In addition, he thought petitioner might have an unsuspected psychiatric diagnosis. Id. Petitioner had a trace neurologic finding localized to the right cerebellum or brainstem given her rotary nystagmus and hypotonia. Id. at 786-87. He wanted to review her brain MRI scan. Id. at 787. He also planned to have a transcranial duplex, a vascular study of the blood vessels in the brain to try to recreate the small vessel disease seen on the SPECT scan. Id.

On January 13, 2012, Dr. Lyden followed up with additional information. Id. He did a physical examination and commented that her mental status was again remarkable for a significant indifference to her medical state. Id. at 787-88. She spoke in a foreign accent for half the visit. Id. at 788. The foreign accent syndrome resolved spontaneously and she finished her visit with her midwestern American accent. She did not have any change in her cranial nerve exam, motor or sensory function or reflexes. He noted that in petitioner's tilt-table test, she had an acute onset of dysarthria and dystonic posturing. Multiple brain MRI scans were normal. She took Norpace for possible dysautonomia from a post-vaccine reaction. No evidence in the record supported a diagnosis of GBS. No evidence supported the diagnosis of hypotension. However, there were repeated descriptions of bizarre and unusual symptoms related to postural changes. Id. She had a significant dystonic and syncopal reaction during a carotid ultrasound. Id. at 789.

Dr. Lyden's overall impression was that petitioner's transcranial doppler study was essentially normal. Id. He recognized that she embellished her symptoms although she did have rhabdomyolysis during an episode of dehydration. She had symptoms suggestive of dysautonomia despite the negative tilt-table test. He thought a good psychologist should evaluate petitioner. Id. To Dr. Lancaster, Dr. Lyden favored a psychogenic etiology for her symptoms and he changed his initial impressions once he had additional data. Id. at 790.

Dr. Lancaster discussed Dr. Yan-Go's records. Id. He described her as a respected expert in autonomic neurologic disorders. She performed a careful evaluation looking for objective evidence of autonomic dysfunction in petitioner. After weighing that evidence, Dr. Yan-Go concluded petitioner did not have a significant autonomic dysfunction. Id. Dr. Yan-Go specifically stated that petitioner had a very complex symptomatology, many symptoms of which she could not explain with a unified disorder. Id. at 791. Dr. Lancaster stated that means Dr. Yan-Go could not find one explanation for all of petitioner's symptoms. Dr. Lancaster said Dr. Yan-Go suspected petitioner had a functional component. "Functional" means psychogenic. Id.

On September 14, 2010, Dr. Yan-Go wrote she still thought overall petitioner did not have any serious pure autonomic failure or degenerative dysautonomia. Id. Dr. Yan-Go wanted to treat petitioner symptomatically and make sure petitioner did not become deconditioned. Id. at 792. Dr. Yan-Go wanted to prevent petitioner having subconscious brain patterning that would affect her and lead to further disability. This was approximately one year after petitioner's flu vaccination. Dr. Lancaster mentioned that Dr. Yan-Go is eminently qualified to detect autonomic dysfunction. Id.

On August 26, 2010, Dr. Kevin Ghassemi, a gastroenterologist, notes petitioner's complaints of vomiting and difficulty swallowing. Id. at 794. He notes petitioner can eat only if she exercises first. Id. Dr. Lancaster said if petitioner were having effects on the cardiovascular part of her autonomic nervous system, exercise would make everything worse, not better. Id. at 795. Exercise stresses and uses the cardiovascular autonomic system to maintain one's blood pressure. Id. Dr. Lancaster stated that the idea that intense exercise would somehow solve a gastrointestinal dysautonomia does not make a lot of sense, particularly that petitioner would, as she told Dr. Ghassemi, have a 24-hour benefit from exercise. Id. at 796. Dr. Ghassemi wrote that petitioner was unlikely to have a primary gastrointestinal motility disorder. Id. at 797.

Dr. Ghassemi had petitioner undergo an upper GI series and esophageal manometry, the first outlining what is going through the digestive tract and the second measuring pressure. Id. at 798. Both studies were basically normal. Dr. Ghassemi deferred to neurologists as to any neurologic diagnosis of petitioner. As for making a gastrointestinal diagnosis, he concluded she did not have evidence of esophageal dysmotility. Id.

On March 30, 2012, petitioner underwent a gastric emptying study. Id. at 799. It showed markedly prolonged gastric emptying consistent with gastroparesis. Id. at 799-800. This is two and one-half years post-vaccination. Id. at 800. Dr. Lancaster said there was a major confounding factor at that time which was petitioner was taking medications, some of which can interfere with gastric motility, particularly Sandostatin, which is known to prolong gastric emptying. Dr. Lancaster said Sandostatin could be why petitioner's stomach emptying was slow. Id. To Dr. Lancaster, this one test of gastric emptying does not in the overall context of the case make it probable that petitioner has autonomic failure. Id. at 801. Dr. Lancaster said that gastroparesis is not itself a disease, but a finding, that might have numerous causes including medications. Id. at 803.

Dr. Lancaster's opinion is that petitioner may have gastroparesis due to medications, but she did not have gastrointestinal dysautonomic disorder in light of multiple other tests. Id. at 808. Since petitioner's vaccination was two or three years earlier, Dr. Lancaster does not think anyone can plausibly link it to the vaccine. Tr. at 809.

Dr. Lancaster does not believe that petitioner's gastrointestinal postprandial hypotension study on October 15, 2010 supports a diagnosis of postprandial hypotension. Id. There were multiple blood pressure readings from 8:15 to 9:35 a.m. and multiple pulse readings over the same period of time. Id. at 809-10. The pulse ranged from 98 to 114. Id. at 810. The systolic blood pressure ranged from 117 to 135 and the diastolic from 77 to 89. All were well within the

range of normal. He thought it would be interesting to know if petitioner developed any symptoms at 45 minutes when she experienced an 18-point drop. He said a healthy, athletic young woman with a blood pressure of 117/82 and a pulse of 108 should be feeling fine. He thinks it would be a big mistake to regard this drop as evidence of postprandial hypotension as an explanation for her symptoms. Id.

Dr. Lancaster stated that Dr. Cintron's diagnostic impression shifted from 2009 going into 2010 and beyond. Id. at 814. Initially, Dr. Cintron wrote about dystonia or myoclonic features, i.e., abnormal postures and jerking. Id. Later, he began to suspect an autonomic disorder or gastroparesis. Id. at 814-15. Dr. Lancaster does not see convincing evidence of autonomic dysfunction in Dr. Cintron's records. Id. at 815. From 2010, Dr. Cintron considered the diagnosis of dystonia or a dystonic-like illness. Dr. Lancaster disagrees with this diagnosis based on seeing the videos. Other neurologists did not diagnose petitioner with dystonia. In 2010 and afterward, the symptoms upon which Dr. Cintron relied to diagnose dystonia became much less prominent. Id.

Dr. Daniel V. Wilkinson, Jr., a cardiologist, saw petitioner on December 15, 2010 and took a history from petitioner in which she said she had POTS. Id. at 816. Dr. Lancaster stated that Dr. Wilkinson did not arrive at the diagnosis of POTS independently. Id. at 817. Dr. Wilkinson's note clarifies that petitioner began taking Sandostatin well before she underwent the gastric emptying test. Id. Dr. Wilkinson notes that petitioner manifested multiple very unusual symptoms, including changes in her speech and language patterns associated with a presumed drop in blood pressure. Id. at 818. Dr. Wilkinson notes petitioner's exercise tolerance is poor. She told him walking up two flights of stairs will cause her to lose speech and become presyncopal. Id. She reported episodic chest pain. Id. at 819. This went away with exercise and she felt best when she exercised vigorously. Dr. Lancaster comments that it makes no sense to write petitioner had poor exercise tolerance when she said she feels best when exercising vigorously. Dr. Lancaster thinks this is why Dr. Wilkinson said petitioner's symptoms were very unusual. Dr. Lancaster thinks that petitioner's difficulty in climbing two flights of stairs would be much worse if she exercised vigorously. It would cause more disabling symptoms. Id. Dr. Lancaster said it is not easy to explain this with any organic disease of the heart or the neurologic system for the autonomic nervous system. Id. at 820.

Dr. Lancaster explained that one of the many factors forming his opinion that autonomic dysfunction is not the correct diagnosis (and myasthenia gravis would not cause these complaints either) is that someone with either autonomic dysfunction or myasthenia gravis would get much worse with exercise. Id.

Dr. Blair Grubb, on July 9, 2011 evaluated petitioner who told him she had GBS after flu vaccination and as a reaction to it. Id. at 820-21. She then had extreme hypotension and also rhabdomyolysis. Id. at 821. Dr. Lancaster said the history petitioner gave Dr. Grubb is not accurate compared to the contemporaneous medical records. Id. at 822. First, she said that she was diagnosed with GBS which her treating doctors ruled out. Dr. Lancaster said the diagnosis of GBS would be important to explain autonomic dysfunction in regulating her blood pressure. Id. Any doctor would think autonomic dysfunction was a logical result of having GBS. Id. at

823. Dr. Lancaster noted petitioner was hypertensive at this time with her blood pressure measuring 148/78. When petitioner stood, her blood pressure did not drop but was 148/84. Thus, petitioner did not show any evidence of having autonomic failure when she saw Dr. Grubb. To answer why a healthy, athletic young runner had blood pressure that was too high, Dr. Lancaster answered she was on Midodrine, a medication used to increase blood pressure in patients who are suspected of having autonomic failure. Dr. Lancaster said the medicine probably caused petitioner to be hypertensive. Id.

Dr. Lancaster disagreed with Dr. Grubb's assessment of petitioner's condition as an autonomic neuropathy due to a vaccine reaction because petitioner gave him incorrect information. Id. at 824. Moreover, petitioner did not have postural hypotension when she saw Dr. Grubb. She was hypertensive. Id. Dr. Lancaster noted that petitioner had a markedly different recollection during her testimony of some of the visits compared to what the doctors documented. Id. at 826. He said this is extremely common in conversion disorder. Id.

Dr. Joey Gee worked petitioner up on May 17, 2012 for myasthenia gravis. Id. at 828. Dr. Gee recorded a myriad of symptoms relating to dysautonomia, weakness, and possible vasculopathy, and records that petitioner has seen rheumatologists, cardiovascular specialists, neurologists, cognitive specialists, orthopedists, ophthalmologists, and other specialists. Id. Dr. Gee notes quite correctly that myasthenia gravis is a neuromuscular junction process and could account for petitioner's weakness, but it would not account for cerebral changes with a vasculitis pattern or for optic neuritis. Id. at 828-29. Dr. Gee considers GBS and chronic inflammatory demyelinating polyneuropathy ("CIDP") which could induce and promote weakness and dysautonomia. Id. at 829. Dr. Gee notes petitioner had never undergone an EMG and plans to order one for her. This would provide evidence for myasthenia gravis as would doing the repetitive nerve stimulation test. Id. He also planned to do a test for MuSK antibody and he did a test for acetylcholine receptor antibody. Id. at 830.

Dr. Lancaster remarked that petitioner never had optic neuritis. Id. Dr. Lancaster points out how multifocal the symptoms are about which petitioner complained. Id. at 831. Dr. Lancaster said petitioner "bombarded" Dr. Gee with a great deal of information which he, to his credit, tried to decipher. Id. at 832. She also had foreign accent syndrome. She complained of weakness and foot drop. She gave a history of nystagmus, which means abnormal shaking movements of the eyes. She said her arms were weak and she had urinary frequency and dizziness. Dr. Lancaster thinks Dr. Gee was thrown off by the history petitioner gave him of optic neuritis, which led Dr. Gee to consider neuromyelitis optica and multiple sclerosis. Id. Dr. Lancaster said:

> I feel what Dr. Gee is going through here. There was just a tidal wave of information that came in and many things that do not appear to be accurate were given to him.

Id. at 832-33.

Petitioner gave Dr. Gee a new symptom of post-exertional headaches and a new symptom of foot drop. Id. at 833. Dr. Lancaster said Dr. Gee did not find an organic basis for these

symptoms. Dr. Gee considered whether petitioner had vasculitis because that could damage multiple different parts of the nervous system, causing tiny strokes to different areas of the brain, spinal cord, and peripheral nerves. The SPECT study would have thrown him off and made him worry about vasculopathy. Dr. Lancaster said Dr. Gee was understandably thinking about "a ton of different diseases under this avalanche of information" that petitioner gave him. Id. Dr. Lancaster said the SPECT study can be inconsistent and not specific to individual neurologic diagnoses. Id. at 835. The fact that the subsequent PET study was normal makes Dr. Lancaster consider the SPECT result a false positive. Id.

Dr. Lancaster said that no doctor diagnosed petitioner with vasculitis and he does not believe she had vasculitis. Id. at 836. Vasculitis means strokes of different tissues. Brain vasculitis causes permanent severe brain injuries. Vasculitis of the peripheral nerves generally causes permanent very severe nerve damage that objective evidence would find. Id.

The results of the testing Dr. Gee ordered were negative for myasthenia gravis and the PET study was normal. Id. Dr. Sheean in San Diego tested petitioner a second time for myasthenia gravis on January 29, 2015. Id. at 837. Dr. Sheean's testing was to quantify levels of different antibodies in petitioner's blood. All the tests were normal. She had normal IgA, SSA and SSB (which test for lupus-like conditions), and acetylcholine receptor antibody (which was zero, which is as negative as it could possibly be since normal is less than 45). Id. at 837. Dr. Lancaster explained there are two different kinds of acetylcholine receptor antibody test. One is for antibodies that modulate the receptor and the other is for antibodies that block the receptor. Id. Petitioner was negative for both kinds of acetylcholine receptor antibodies. Id. at 838. Petitioner was tested for voltage-gated calcium channel antibodies which, if the test were positive, would support a diagnosis of Lambert-Eaton syndrome. That, too, was negative. She was tested for antibodies to the voltage-gated channel complex, which was negative. Id.

The undersigned asked Dr. Lancaster why Dr. Sheean diagnosed petitioner with myasthenia gravis when all her test results were negative, a result that was consistent with the results of the tests Dr. Gee ordered three years earlier. Id. at 838-39. Dr. Lancaster responded that he thinks Dr. Sheean was using myasthenia gravis as a working diagnosis, but he was looking for objective confirmation that his diagnosis was correct. Id. at 839. Petitioner's repetitive stimulation studies were done again and were yet again negative. Id. at 840. As all these tests came back negative, Dr. Lancaster said it was very unlikely that the diagnosis of myasthenia gravis was correct. Id. at 841.

Dr. Lancaster thinks if Dr. Sheean had seen petitioner's videos and Dr. Buttar's records, Dr. Sheean might have had a very different impression of how likely it was that petitioner has myasthenia gravis. Id. Dr. Lancaster wondered if Dr. Sheean would do the one test on petitioner that he did not do: single fiber test. Id. He said that people with conversion disorder who receive treatments for illnesses they do not have are being put at risk. Id. at 843. Dr. Lancaster said he does not have an organic explanation for petitioner's testifying that when Dr. Sheean raised the dosage of her IVIG, her myasthenia gravis symptoms got better, but her autoimmune dysautonomia symptoms got worse. Id. at 844-45.

As for Dr. Steinman's assertion that petitioner's test results were negative because she was on IVIG, Dr. Lancaster said the initial tests were done before she went on IVIG. Id. at 846. He does not think it is true that IVIG would cause a negative result. Id. Dr. Lancaster explained petitioner's statement that IVIG helped her symptoms by saying this was the same placebo effect as her positive responses to Dr. Buttar's treatments. Id. at 846-47. IVIG is an incredibly powerful placebo. Id. at 847. What Dr. Lancaster saw in the hearing room on the second day of trial when petitioner sank to the floor growling was not indicative of a myasthenic crisis. Id. She also had an episode of stridor when her dog and cat were fighting on November 4, 2013. Id. at 848.

Dr. Lancaster remarked that the videos showed petitioner with large flailing movements of her limbs, raising her arms up and down, which he would not associate with a natural dystonic posture of the limb which usually involves a forcible contraction and fixed flexion of the hands. Id. at 853. Patients with organic dystonia probably have a genetic etiology and not an autoimmune etiology in Marsden's article (Ex. 170). Id. For the patients in Marsden's article, the dystonia came on gradually and persisted for years, remaining the same. Id. at 853-54. The patients did not have rapid fluctuation among different symptoms. Id. at 854. They did not have associated symptoms of seizure-like events, passing out, feeling events were triggered by their tongue, feeling cold spots, and feeling profound weakness. Dr. Lancaster, in comparing petitioner with the subjects of Marsden's article, called them as different as night and day. Id.

In the Fahn article (Ex. 129), the subjects had dystonia and used sensory tricks. Id. The patients often developed deformities in the neck from having their neck twisted in one direction over a prolonged and sustained contraction. Id. at 855. Fahn is describing torsion dystonia, a twisting of part of the body, particularly the neck. Mostly the cause is genetic or idiopathic, not considered to be autoimmune. Id. Dr. Lancaster noted that most of petitioner's doctors who observed the prominent movements of her gait, such as Dr. Urrutia, thought it was non-physiologic. Id. at 856. Dr. Lancaster does not think petitioner had any autoimmune neurologic injury in proximity to her flu vaccination. Id. He does not think petitioner had autoimmune autonomic ganglionopathy. Id. at 857. He stated, "I do not believe she had any organic disease induced by vaccination." Id. at 858.

Dr. Lancaster took issue with Dr. Steinman's thesis that there is molecular mimicry between flu virus and myelin proteins, eliciting nervous system inflammation leading to autoimmune dysautonomia. Id. at 858, 859. Dr. Lancaster stated:

> Myelin is ubiquitously expressed in myelinated fibers of the nervous system. The idea of a selective attack against a myelin antigen in such a way as to only damage the autonomic parts of the nervous system without damaging the myelin to the arms and legs, while … also ignoring … myelin in the optic nerve, in the brain, that's very unlikely. I don't believe any disease like that has been shown to exist.

> And, so, one of the things about this case is we're not just saying that the vaccine caused Petitioner to get some well-established disease, such as Guillain-Barre syndrome. . . . [Petitioner] has a novel disease process that's not an accepted neurological disease. . . . [T]here is no precedent for it.

Tr. at 859.

He continued:

> In the case of myasthenia gravis, we are talking about a disease that we all agree absolutely exists and whether or not she has it. When we're talking about whether or not she has an autoimmune disease selective to myelin of pre-ganglionic fibers in the autonomic nervous system only, then I don't know what disease we're even talking about. It's not like we can consult a large literature on that disease and what its diagnostic criteria are, because there isn't one.
>
> So, that's a very different thing from debating whether someone has or doesn't have a known disease, as we're discussing a novel disease.

Id. at 860-61.

Dr. Lancaster's testimony prompted questions from the undersigned and the following colloquy took place:

> THE COURT: All right. Tell me, because this is the first time I've heard this kind of comment, is what Dr. Steinman has been identifying with various terms as an autonomic autoimmune neuropathy, does that disease entity not exist?
>
> THE WITNESS. No. So, the disease entity of autoimmune autonomic ganglionopathy, autoimmune autonomic neuropathy exists. [T]he one mechanism that's been established for approximately half of those patients is autoantibodies to the ganglion acetylcholine receptor. Absolutely exists. I don't think Petitioner had it. [B]ut this idea that at times has been mentioned as a potential disease mechanism of autoimmunity selectively to myelin in the autonomic nervous system, I don't know what disease that is.
>
> THE COURT: So, not only does the physiologic description not make sense, but the disease that would manifest if it existed, doesn't exist?

> THE WITNESS: Well, it's never been shown to exist as a disease mechanism, in any person. So, it's one thing to say that someone has a vaccine causing Guillain-Barre syndrome. [He or she] had demyelination of [his or her] autonomic nervous system along with other things. I'm not aware of any evidence, for instance, in patients with autoimmune autonomic neuropathy that has anything to do with demyelination of the autonomic nervous system. That's an idea. I'm not aware of any patient where that's been proven to be the case in that sort of selective autonomic neuropathy. The ones that we understand well are autoantibodies to ganglionic acetylcholine receptors, which is a different thing. [I]t's analogous to myasthenia gravis. …
>
> THE COURT: [I]f you don't have GBS and you don't have CIDP and all you're asserting is you have autonomic symptoms which are somehow autoimmune because there's myelin in autonomic nerves, that just doesn't exist by itself?
>
> THE WITNESS: As far as I know, that has not been shown to exist.

Id. at 861-63.

Dr. Lancaster said that autoimmunity to myelin in the brain would cause multiple sclerosis and acute disseminated encephalomyelitis. Id. at 864. Autoimmunity to peripheral nerves causes GBS or CIDP. Id. Myelin proteins have not been implicated in the pathophysiology of autoimmune autonomic neuropathy. Id. at 866. Dr. Lancaster testified that there is no logical or scientific connection of myelin proteins and the pathophysiology of dystonia. Myelin proteins have not been implicated in the pathophysiology of myasthenia gravis either. Id.

All these illnesses have different loci. Id. at 867. Myasthenia gravis localizes to the neuromuscular junctions on skeletal muscle cells to particular proteins on those skeletal muscle cells in the neuromuscular junction that are used to receive signals or organize receptors. Autoimmune autonomic neuropathy involves the sympathetic and parasympathetic parts of the peripheral nervous system. Dystonia comes from the brain. Petitioner has many other symptoms that cannot be explained by all these diseases. Dr. Lancaster said Dr. Steinman has not provided a reliable explanation for flu vaccine causing petitioner this constellation of symptoms. Id.

Dr. Lancaster said that if we were going to attribute petitioner's illness to flu vaccine, we need a plausible explanation of what was wrong with petitioner's gait and her speech, and why she was having seizure-like episodes, cold spots, abnormal sensations, tongue paralysis and numerous other symptoms. Id. at 868. He said, "We cannot just ignore her symptoms and decide to explain other symptoms that became much more prominent later because that's more easy to explain." Id.

Dr. Lancaster thinks infection superimposed on exercise is perfectly reasonable and the most likely explanation for petitioner's rhabdomyolysis. Id. There is a clear, contemporaneous record of petitioner having cold-like symptoms, sore throat, subjective fevers, and green phlegm to assume she had a cold at the time. Id. at 869.

(Because respondent's other expert, Dr. Whitton, had a plane to catch, the undersigned took his direct and cross examinations before Dr. Lancaster's cross examination. But for the sake of consistency, the undersigned summarizes Dr. Lancaster's cross examination first below.)

On cross-examination, Dr. Lancaster said this case was the third or fourth one in which he testified. Id. at 994. He has testified in any kind of court 12 or 13 times. In his practice, he has made the diagnosis in 100 patients of some form of conversion disorder over a 13-year period. Id. at 998. For neurology in general, that's very typical. For someone who works in epilepsy, that would be a huge underestimation. Id. It would also be a huge underestimation for someone who works in movement disorders. Id. at 999. Dr. Lancaster's own clinic focuses on autoimmune encephalitis for which there is abundant evidence of organic disease. That is why the number of patients with conversion disorder is lower. Id. Dr. Lancaster said petitioner's rhabdomyolysis was relatively mild and short-lived. Id. at 1024. It was essentially gone by the time she had her most severe symptoms when she went to Dr. Buttar's clinic. At that time, Dr. Lancaster did not observe any signs of physiological illness. Petitioner was in great physical condition, running an 8K race in a pretty good time, performing multiple highly strenuous gaits which are on the videos, and numerous activities requiring good overall physical health to perform that astasia-abasia gait. Dr. Lancaster did not observe any physiologic illness that triggered what was occurring. Id. Dr. Lancaster said he does not know of any evidence for rhabdomyolysis causing conversion disorder, particularly when it is self-limited and already over. Id. at 1025.

Dr. Lancaster said cold-like syndromes are caused by hundreds if not thousands of different viruses and families of viruses. Id. at 1029. He said there is no comprehensive viral test panel that can exclude a viral infection. Id. Petitioner on September 18, 2009 had tachycardia in the context of shortness of breath, a cough producing sputum, and difficulty breathing, which Dr. Lancaster said was extremely common in anyone who his sick with a cold or other illness. Id. at 1044, 1047. That indicates petitioner's autonomic nervous system was working, not that it was not working. Id. at 1047. Dr. Lancaster said that he did not observe any actual syncope petitioner was having despite seeing many syncope-like events. Id. at 1052.

Dr. Lancaster said Dr. Cintron was missing vital information when he diagnosed petitioner. Id. at 1055. Dr. Cintron was missing the Johns Hopkins Hospital medical records. Id. In none of Dr. Cintron's notes did Dr. Lancaster see a good discussion of petitioner's seizure-like events in assessing whether they were seizures or non-epileptic seizures. Id. at 1056.

Dr. Lancaster said that POTS is a clinical syndrome that is very loosely and problematically defined. Id. at 1089. He has diagnosed several patients with autonomic disorders but, in general, he does not diagnose people with POTS. Id.

On redirect, Dr. Lancaster said that petitioner's antibodies were never tested for acetylcholine receptors. Id. at 1099. In his opinion, she does not have autoimmune autonomic neuropathy or autoimmune autonomic ganglionopathy. Id. Petitioner's minimal heart murmur is due to a structural issue and not due to an autonomic disorder. Id. at 1104.

Dr. Whitton's Testimony

Respondent's second expert, Dr. James L. Whitton, testified. Id. at 872. He works at the Scripps Research Institute in La Jolla, California. This is not the same Scripps as Scripps Health. Id. Scripps Research Institute is a large nonprofit research institute of independent investigators. Id. at 872-73. Dr. Whitton has been there 31 years. Id. at 873. Dr. Whitton is in the department of immunology and microbial science. Id. He has studied vaccines extensively. Id. at 874. He has also studied the immune response to viral infection. Id. He studies how vaccines impact the immune system. Id. at 874. Dr. Whitton has a medical degree but is not a licensed medical doctor and not licensed to practice medicine in the United States. Id. at 875. He also has a Ph.D. Id. He does not treat patients. Id. at 876. Dr. Whitton's role in this case is to comment on the mechanisms Dr. Steinman proposed for the cause of disease. Part of Dr. Whitton's lab work focuses on how viruses cause disease, rather than looking at the immune response. Id.

Recognizing that Dr. Steinman's opinion evolved over the reports and during the hearing, Dr. Whitton said that two conditions at issue are rhabdomyolysis and autoimmune dysautonomia. Id. at 880. There is also the issue of myasthenia gravis. Dr. Whitton describes Dr. Steinman's theory as flu vaccine inducing an immune response that cross-reacted with petitioner's myelin. Id. Dr. Steinman's argument is that flu vaccine components attacked the preganglionic fibers of petitioner's autonomic nervous system. Id. at 881. Dr. Steinman's thesis is that destruction of the myelin on these preganglionic fibers causes autonomic dysfunction. Dr. Whitton was surprised that one of the autonomic symptoms Dr. Steinman described as being affected included walking. Id.

Dr. Whitton described molecular mimicry as an immune reaction of a host protein to a viral or bacterial protein. Id. at 882. The two proteins have to be sufficiently different to trigger an immune response, but sufficiently similar so that response, once triggered, can attack the host protein. Id. at 882-83. An example of molecular mimicry is the use of rabies vaccine causing neuroparalysis among vaccinees. Id. at 883. The rabies vaccine was prepared in animal brains. Id. at 884. Roughly 0.5 percent of vaccinees developed encephalitis. Id. at 884-85. Dr. Whitton said molecular mimicry is not easy to trigger because with rabies vaccine including both animal brain protein and rabies protein, and with multiple injections, only 0.5 percent of people developed encephalitis. Id. at 887. He said this vaccine had quite a bit of homology since the central nervous system proteins of sheep and the central nervous system proteins of humans have a lot of homology. "Sequence homology" means at the level of individual amino acids. Id.

Dr. Whitton said Dr. Steinman discussed the sequence FFKN present in myelin basic protein and compared that to FYKN present in some strains of flu virus. Id. at 888. When one compares FFKN to FYKN, there is no identity. Id. Dr. Steinman proffered four theories, the first two based on myelin basic protein. Id. at 891. The first theory could be called the myelin

basic protein antibody theory. The second theory could be called the myelin basic protein T-cell theory. Both theories rely on homology or substantial cross-reactivity between a vaccine sequence that petitioner did not receive because Dr. Steinman picked a subsequent flu vaccine season flu vaccine. Because of Dr. Steinman's mistaken analysis of the incorrect flu vaccine, Dr. Whitton considers those first two theories out of the discussion. The third theory is a sequence in the Brisbane component of the flu vaccine triggered a cross-reactive response that could attack one of two different myelin proteins. Id. One is called MOG and the other is called CNPase. Id. at 891-92. Dr. Whitton did a search of A/Brisbane/10/2007 and discovered it is not an immune epitope capable of triggering an immune response. Id. at 892. He said no one has ever shown that the Brisbane H_3N_2 virus induces a T-cell response. Id.

Dr. Whitton said that Dr. Steinman was no longer talking about myelin basic protein but MOG or a component of myelin called myelin oligodendrocyte glycoprotein for which autoimmune responses to MOG in animal models indicate the autoimmune damage tends to be more restricted to the central nervous system than to the peripheral nervous system. Id. at 895. Dr. Steinman's thesis is that there is a very highly specific attack on either MOG or CNPase in this subset of peripheral nerve cells on the preganglionic fibers. Id. He uses the Markovic-Plese article (Ex. 152) as proof of his thesis. Id. at 897-88. The subject that the authors studied had MS and an acute active flu A virus infection. Id. at 899. The authors took blood from the patient and made a T-cell clone. Id. The authors then incubated the mixture of T-cells with a specific peptide that represents a viral epitope. The sequence of the epitope was PKYVKQNT-KLAT. Id. The authors asked themselves what other peptide sequences might stimulate the T-cell clone. Id. at 900. Flu A viruses all stimulated the clone. Id. at 901. The authors took four peptides: (1) the viral peptide; (2) and (3) oligodendrocyte myelin glycoproteins ("MOG"); and (4) CNPase. Id. at 902. The viral peptide was the positive control. Id. at 902-03. The authors stimulated the T-cell clone with either no peptide or a lot of peptide. Id. As they increased the peptide dose, they increased the division of the T-cells. Id. The two MOG peptides stimulated the T-cell clone a little bit. Id. at 904. The best stimulation came from the CNPase peptide. Id.

Dr. Whitton said that a researcher doing synthetic peptide stimulation adds billions of peptides to the well. Id. at 905. That opens the experiment to artifacts. Id. Research with actual, not synthetic, peptide sequences can be very different. Id. at 906. Going back to the Markovic-Plese article, Dr. Whitton pointed out that the sequence of the CNPase, which is LYSLGNGRWN, when compared to the viral sequence, which is UVKNTLKLE, is not very homologous. Id. at 908. The authors state the same conclusion on page 37 (internally marked page 7) of their paper: the CNPase-derived peptide had no homology with the native flu HA epitope. Id.

Dr. Whitton asked what is the evidence that a T-cell might be pathogenic. Id. at 908. Dr. Steinman is making many presumptions. Dr. Whitton does not see evidence that the vaccine induces a T-cell that could cross-react with MOG or CNPase. But if it did, does that mean the T-cell is pathogenic? Id. Dr. Whitton said a T-cell might not be the cause of an illness but the result of it. Id. at 909. Dr. Whitton does not think flu vaccine induces pathogenic T-cells that

attack a very specific place in the peripheral nervous system. Id. at 910. He would call a T-cell or an antibody autoimmune only if it causes disease. Id. at 911.

Dr. Whitton described Dr. Steinman's fourth theory that flu virus triggers the induction in a vaccinated or infected host of antiganglioside antibodies which then attack gangliosides which are components of myelin. Tr. at 912. Dr. Whitton notes that antiganglioside antibodies have been implicated in GBS particularly GBS that Campylobacter jejuni causes. Id. Dr. Steinman testified that this theory was his most important theory, although it only appeared in Dr. Steinman's fifth expert report. Id. at 913. Dr. Whitton turned to the Nachamkin article (Ex. LLL and Ex. 146). Id. at 914. The article is based on the observation of the increase in number of GBS cases among swine flu vaccine recipients compared to the number of baseline GBS cases in people not vaccinated. Id. The authors found that the swine flu vaccine induced antiganglioside antibodies, but also found that additional flu vaccines not associated with an increased incidence of GBS also induced anti-ganglioside antibodies, as well as IgM and IgG antibodies in mice. Id. at 916.

Dr. Whitton turned his attention to the Lei article (Ex. OOO). Id. at 918. The study's aim was to find out if the 2009 H_1N_1 vaccine induced antiganglioside antibodies in humans and mice. Eight patients had post-vaccination GBS after receiving this vaccine. Id. The authors did not detect antibodies against ganglioside in any of these people or in vaccinated mice. Id. at 918-19. The authors conclude their study results do not support the proposition that flu viruses or flu vaccines induce antiganglioside antibodies. Id. at 919.

Dr. Whitton explained that flu viruses have a receptor called hemagglutinin which binds to the cell the flu virus wants to infect, and the molecule called sialic acid facilitates cell entry. Once the virus gets into the cell, it replicates. The virus then exits the cell to infect other cells. Id. Neuraminidase is an enzyme in the virus that cleans the sialic acid, releasing the virus from the infected cell. Id. at 919-20. But the virus trails little pieces of the host cell as it escapes from the cell. Id. at 920. The human or mouse forms antibodies to the little pieces of the host cell. That the virus is grown in eggs means that the pattern of glycosylation, i.e., the types of sugars (the sialic acids) might slightly differ from mammalian cells, which increases the chance of inducing antiganglioside antibodies in a human. Dr. Whitton notes that the authors of this study specifically attempted to confirm the Nachamkin article results and could not confirm them. Id.

Because of the Nachamkin and Lei articles, Dr. Whitton does not believe petitioner's flu vaccination induced antiganglioside antibodies. Id. at 930. These two articles do not support Dr. Steinman's thesis that flu vaccine induced in petitioner an antiganglioside antibody response. Id. at 931. He also does not believe that the flu vaccine petitioner received induced a T-cell response to MOG or CNPase. Id. Dr. Whitton said that antibodies are not associated with rhabdomyolysis. Id. at 932. He thinks the cause of petitioner's rhabdomyolysis is much more likely to be either petitioner's concurrent viral infection or exercise or both. Id. at 932-33. He does not think that flu vaccine caused petitioner's rhabdomyolysis. Id. at 933.

As for autoimmune autonomic neuropathy, Dr. Whitton testified there is no evidence that flu vaccine can cause an antibody response against the acetylcholine receptor. Id. Dr. Whitton

recalled that once Dr. Steinman learned at the hearing that he had analyzed the wrong flu vaccine (2010-2011 instead of 2009-2010), Dr. Steinman said he could not link through homology flu vaccine with acetylcholine receptor because there is no known homology. Id. at 934. Dr. Whitton said he disagrees with the theory that flu vaccine induces an immune response against the myelin in the preganglionic fibers of the autonomic nervous system to cause autoimmune autonomic neuropathy. Id. at 235. Dr. Whitton does not accept Dr. Steinman's theory that flu vaccine caused an anti-myelin response in the peripheral nerves which innervate muscles and caused myasthenia gravis. Id.

Dr. Whitton said it is very common to have autoreactive antibodies that do not cause disease in healthy people. Id. at 936-37. Even if someone has antiganglioside antibodies, one cannot conclude that induces pathogenic disease. Id. at 937. Dr. Whitton's opinion is that petitioner's flu vaccination did not cause any of her alleged injuries. Id.

On cross-examination, Dr. Whitton explained that to cause disease, a scientist does not want total homology but something close to it. Id. at 947. He accepts that molecular mimicry is mainstream science. Id. at 961. The Markovic-Plese article states the protein sequence is not homologous at the amino acid level. Id. The CNPase-derived peptide had no homology with the native flu HA epitope. Id. at 962. Markovic-Plese does not show a reaction to vaccine. Id. at 971. It shows a reaction to a peptide taken from a vaccine. Id.

On redirect, Dr. Whitton said that muscle cells are not myelinated. Id. at 988. Muscle cells are innervated by myelinated nerves. Id. GBS can cause rhabdomyolysis and neurogenic problems with muscles, inducing creatine kinase. Id. at 989. "Innervated" means they have a nerve cell connection. The electricity changes into chemistry at the synapse and the electricity is restored at the level of the myocyte (the muscle cell). Id. In other words, "innervation" means stimulus. Id.

DISCUSSION

To prevail under the Vaccine Act, petitioner must prove by preponderant evidence that a vaccination caused her injury. 42 U.S.C. §§ 300aa-11(c)(1), -13(a)(1)(A). If petitioner's alleged injury satisfies the criteria of being a Table injury, the Act presumes causation. 42 U.S.C. § 300aa-11(c)(1)(C)(i); Broekelschen v. Sec'y of HHS, 618 F.3d 1339, 1341-42 (Fed. Cir. 2010); Andreu v. Sec'y of HHS, 569 F.3d 1367, 1374 (Fed. Cir. 2009). Where, as in the instant action, petitioner's alleged injuries are not Table injuries, she must prove causation in fact. Broekelschen, 618 F.3d at 1342 (citing Moberly ex rel. Moberly v. Sec'y of HHS, 592 F.3d 1315, 1321 (Fed. Cir. 2010)). To prevail in a causation-in-fact case, "petitioner must show that the vaccine was 'not only a but-for cause of the injury but also a substantial factor in bringing about the injury.'" Stone v. Sec'y of HHS, 676 F.3d 1373, 1379 (Fed. Cir. 2012) (quoting Shyface v. Sec'y of HHS, 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)). "Once petitioner has demonstrated causation, she is entitled to compensation unless the government can show by a preponderance of the evidence that the injury is due to factors unrelated to the vaccine." Broekelschen, 618 F.3d at 1342 (citing Doe v. Sec'y of HHS, 601 F.3d 1349, 1351 (Fed. Cir. 2010); 42 U.S.C. § 300aa-13(a)(1)(B)).

To satisfy her burden of proving causation in fact, petitioner must prove by preponderant evidence: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." Althen v. Sec'y of HHS, 418 F.3d 1274, 1278 (Fed. Cir. 2005). In Althen, the Federal Circuit quoted its opinion in Grant v. Secretary of Health and Human Services, 956 F.2d 1144, 1148 (Fed. Cir. 1992):

> A persuasive medical theory is demonstrated by "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury [,]" the logical sequence being supported by a "reputable medical or scientific explanation[,]" i.e., "evidence in the form of scientific studies or expert medical testimony[.]"

418 F.3d at 1278.

Without more, "evidence showing an absence of other causes does not meet petitioner's affirmative duty to show actual or legal causation." Grant, 956 F.2d at 1149. Mere temporal association is not sufficient to prove causation in fact. Id. at 1148.

The Federal Circuit has held that petitioner must present more than a possible causal link between vaccination and injury; moreover, the causal link must be based upon persuasive and reputable evidence. Paterek v. Sec'y of HHS, 527 F. App'x 875, 879 (Fed. Cir. 2013).

The Federal Circuit in Capizzano v. Secretary of Health and Human Services, 440 F.3d 1317, 1326 (Fed. Cir. 2006), emphasized that special masters are to evaluate seriously the opinions of petitioner's treating doctors since "treating physicians are likely to be in the best position to determine whether a logical sequence of cause and effect show[s] that the vaccination was the reason for the injury." See also Broekelschen, 618 F.3d at, 1347; Andreu v. Sec'y of HHS, 569 F.3d 1367,1375 (Fed. Cir. 2009). Dr. Steinman calls petitioner's treating doctors "boots on the ground." Petitioner has seen and/or had tests interpreted by 56 doctors[163] and two psychologists,[164] and has visited 11 medical institutions.[165] The undersigned has evaluated

[163] Dr. Zachary Malachias, Dr. Brian A. Hazen, Dr. Pranav Vermani, Dr. Sarbjot S. Dulai, Dr. Ho-Song Lee, Dr. Jeffrey S. Luy, Dr. Michael Rodriguez, Dr. Sarfraz A. Choudhary, Dr. Scott Weir, Dr. Mohammed A. Mannan, Dr. Elise Berman, Dr. Jonathan Bresner, Dr. Paul M. Dellemonache, Dr. Garry Ho, Dr. Julius C. Pham, Dr. Anjail Sharrief, Dr. Victor C. Urrutia, Dr. Christopher C. Oakley, Dr. Ruben Cintron, Dr. Rashid Buttar, Dr. Randolph R. Stephenson, Dr. Mark P. Tanenbaum, Dr. Nick Cossa, Dr. David Kruse, Dr. Pradeep Nayak, Dr. Arun Kumar, Dr. Walter Atiga, Dr. Stuart A. Fruman, Dr. Farhad Zangeneh, Dr. Neil Q. Tran, Dr. Frisca Yan-Go, Dr. Kevin Ghassemi, Dr. Sheri Hart, Dr. Robert A. Wohlman, Dr. Daniel V. Wilkinson, Jr., Dr. Blair Grubb, Dr. Kelly E. Williams, Dr. Alan D. Waxman, Dr. Elmer Y. Chang, Dr. Mariko L. Ishimori, Dr. Patrick J. Olek, Dr. Swaraj Bose, Dr. Sam Kipper, Dr. Daniel J. Wallace, Dr. Joey R. Gee, Dr. Jackson W. Penry, Dr. Raj Patel, Dr. Loan T. Nguyen, Dr. Jagmeet S. Mundi, Dr. Geoffrey L. Sheean, Dr. Craig A. Salcido, Dr. Robert Y. Goldberg, Dr. Oscar H. Otanez, Dr. Eric A. Glasser, and Dr. Brian Barry.

[164] Dr. Christine Cosgrove and Dr. Sidney Binks.

[165] Inova Loudon Hospital (twice), Fairfax Hospital, Johns Hopkins Hospital, Reston Hospital, Inova Fair Oaks Hospital, Swedish Medical Center, Toledo Medical Center, Cedars-Sinai Medical Center, Mission Hospital, Mayo Clinic (petitioner did not file any records from the Mayo Clinic but mentioned to Dr. Yan-Go that she had been there), and MedStar Georgetown Hospital.

seriously all the opinions of these 58 doctors and 11 medical institutions in forming an opinion about this case.

Other than there being no dispute that petitioner had rhabdomyolysis in early September 2009, the opinions of these treating physicians fall within three categories: (1) petitioner has conversion disorder and there is nothing physically wrong with her; (2) petitioner has dysautonomia; or (3) petitioner has dysautonomia and conversion disorder. Doctors have also diagnosed petitioner with numerous other medical maladies: gastroparesis, stiff person syndrome, myasthenia gravis, POTS, autoimmune autonomic neuropathy, autoimmune encephalopathy, unspecified GAD65 disorder, and cerebellar ataxia. No doctor has diagnosed petitioner with GBS or optic neuritis although petitioner has given a history to doctors that she had both. Those doctors who had access to petitioner's previous medical records note in their own medical records that she did not have GBS and that her eyes are normal.

Where there is a discrepancy between what petitioner tells her later doctors compared to what the earlier doctors wrote in their medical records, the undersigned believes the earlier medical records when the later doctors base their diagnoses on what petitioner tells them rather than on the earlier medical records. Where there is a discrepancy between what petitioner declared in her filed court statements or testified about in the hearing and the contemporaneous medical records, the undersigned finds more credible the contemporaneous medical records. Well-established case law holds that information in contemporary medical records is more believable than that produced years later at trial. United States v. United States Gypsum Co., 333 U.S. 364, 396 (1948); Burns v. Sec'y of HHS, 3 F.3d 415 (Fed. Cir. 1993); Ware v. Sec'y of HHS, 28 Fed. Cl. 716, 719 (1993); Estate of Arrowood v. Sec'y of HHS, 28 Fed. Cl. 453 (1993); Murphy v. Sec'y of HHS, 23 Cl. Ct. 726, 733 (1991), aff'd, 968 F.2d 1226 (Fed. Cir.), cert. denied sub nom. Murphy v. Sullivan, 113 S. Ct. 263 (1992); Montgomery Coca-Cola Bottling Co. v. United States, 615 F.2d 1318, 1328 (1980). Contemporaneous medical records are considered trustworthy because they contain information necessary to make diagnoses and determine appropriate treatment:

> Medical records, in general, warrant consideration as trustworthy evidence. The records contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium. These records are also generally contemporaneous to the medical events.

Cucuras v. Sec'y of HHS, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

Conversion Disorder

The medical records and/or Dr. Steinman's 10 reports name 21 illnesses or conditions that petitioner has or might have, not all of which she alleges flu vaccine caused: (1) viral infection; (2) rhabdomyolysis; (3) seizures; (4) GBS; (5) dystonia; (6) dysautonomia; (7) autonomic autoimmune neuropathy/autonomic immune ganglionopathy; (8) POTS; (9) neurocardiogenic syncope; (10) vagal nerve damage (11) gastroparesis; (12) myasthenia gravis; (13) stiff person syndrome; (14) optic neuritis; (15) Raynaud's of the brain; (16) cerebral brain flow problems; (17) systemic lupus erythematosus; (18) mercury poisoning; (19) GAD

antibodies-associated syndrome, (20) intestinal pseudo-obstruction; and (21) conversion disorder.

The undersigned starts this discussion with the last condition, conversion disorder. This is respondent's defense of a known factor unrelated to vaccination, 42 U.S.C. § 300aa-13(a)(1). Respondent does not have the burden of proving a known factor unrelated to flu vaccine is the cause in fact of petitioner's symptoms unless petitioner makes a prima facie case that flu vaccine caused at least one of her 20 illnesses and conditions. Respondent also defends that petitioner has failed to make a prima facie case that she has any physical illness other than her transient rhabdomyolysis.

The Federal Circuit held in Broekelschen that determining "causation turns on which injury [petitioner] suffered." 618 F.3d at 1346. The issue in that case was whether flu vaccine caused Dr. Broekelschen transverse myelitis or anterior spinal artery syndrome. Id. at 1342. Dr. Steinman, petitioner's expert in the instant action, was also Dr. Broekelschen's expert. The special master found respondent's neurologic expert more credible than Dr. Steinman, thus dismissing the case. Dr. Broekelschen appealed on the basis that the special master first had to determine if petitioner made a prima facie case of causation in fact and, only then, decide if respondent's known factor unrelated (anterior spinal artery syndrome) was the cause in fact of petitioner's condition. The Federal Circuit disagreed, stating "nearly all the evidence on causation was dependent on the diagnosis of Dr. Broekelschen's injury." Id. at 1346.

The way the instant action has developed is remarkable in that the experts, counsel, and the undersigned have had the unique privilege of seeing contemporaneous videos of petitioner within one month of her flu vaccination when she was on national television broadcasts and visiting Dr. Buttar's clinic in North Carolina. Thus, the experts did not have to rely solely on the contemporaneous medical records to evaluate whether petitioner had a physical illness and, if she did, what was the identity of that illness. Petitioner also had her own website in which she spoke directly to her audience. Interestingly, Dr. Steinman in his testimony said he was more impressed meeting petitioner in person (right before the hearing) than in reading PDFs of her medical records or watching videos on, as he put it, a little screen. His downplaying the importance of those videos is in stark contrast with respondent's expert Dr. Lancaster who called the videos central to the case. The undersigned finds Dr. Lancaster more persuasive about the importance of the videos than Dr. Steinman.

The undersigned relies more on Dr. Lancaster's opinion concerning whether petitioner manifested dystonia, dysautonomia, or autoimmune autonomic neuropathy/autoimmune autonomic ganglionopathy when she was violently flailing her arms and bopping her head up and down while trying to walk a precipitously narrow line forward, but having no problem running forward or walking backward or sideways. The undersigned analyzes the two striking details of these videos, i.e., supposed seizures and purported dystonia affecting petitioner's ability to walk forward and whether these are symptomatic of conversion disorder.

Seizures

The parties might wonder why the undersigned is discussing seizures when, for the first time in this case, petitioner recanted at the hearing that she ever had seizures, using the excuse that she is not a doctor and just assumed the violent bopping of her head up and down was seizures. The reason is the analysis of the videos is central to evaluating the credibility of Dr.

Steinman as an expert in this case. Dr. Lancaster in his first expert report (Ex. H) went painstakingly through each video and described what each video out of 14 hours of videos showed petitioner doing and what his professional opinion of it was. He wrote and testified that petitioner was manifesting psychogenic seizures, in other words, fake seizures. When, a year later, Dr. Steinman wrote his report (Ex. 141) after seeing some of the same videos, his only comment about petitioner's violent bopping of her head was he would need a concomitant EEG and preferably a video EEG in order to comment. Yet, the medical records show that petitioner always had normal EEGs, even when Ms. Preston falsely diagnosed petitioner with an abnormal qEEG. Dr. Cintron, one of petitioner's neurologists who is not only board certified in neurology but also in neuromuscular EMG, reanalyzed the qEEG and then did his own EEG, showing petitioner had a normal EEG.

Dr. Steinman at the hearing was still waffling about whether petitioner had seizures, but he clearly did not pursue the point in his testimony, which when respondent's counsel Ms. Walters whittled it down to its essence was that he believed flu vaccine caused petitioner autoimmune autonomic neuropathy and vagus nerve injury. Seizures are a central nervous system problem and petitioner never had a central nervous system problem. Dr. Steinman agreed that the SPECT scan that purportedly showed cerebral blood flow problems was inaccurate, agreeing with Dr. Lancaster. Moreover, all of petitioner's brain MRIs were normal as were her EEGs. The PET scan of her brain after the falsely positive SPECT scan was also normal.

The undersigned finds that petitioner never had seizures.

Astasia-Abasia

The second notable phenomenon of these videos is crucial to understanding whether petitioner had a physical illness or conversion disorder, i.e., astasia-abasia. Dr. Lancaster wrote in Ex. H and testified that petitioner's narrow, jerky, twisting walk forward with her arms flailing is astasia-abasia, a typical symptom of conversion disorder. He noted that it is actually difficult to walk like that, and, thus, only someone in good physical condition, as petitioner was, can manage it without falling down.

Dr. Lancaster is not alone in noting that petitioner displayed astasia-abasia. Dr. Victor C. Urrutia, a neurologist, noted petitioner had astasia-abasia (med. recs. Ex. 55, at 85). Dr. Randolph R. Stevenson, a neurologist, noted petitioner had astasia-abasia (med. recs. Ex. 22, at 37).

Dr. Steinman likes to use the phrase "boots on the ground" in describing in his expert reports and testimony how important treating doctors' opinions are in forming his own opinion. Yet, not only does Dr. Steinman not consider Dr. Urrutia's and Dr. Stephenson's diagnoses that petitioner had astasia-abasia and, thus, conversion disorder, but also Dr. Steinman never commented once in Ex. 141 or in his testimony about the contemporaneous videos showing petitioner had astasia-abasia and not dystonia. Instead, Dr. Steinman testified that it was only when he saw the videos that he thought petitioner had dystonia. Tr. at 492-93. He reasoned that petitioner could not have known that if she walked backward, she would not have the flailing and jerky walk she had walking forward. Id. at 493. But Dr. Lancaster testified that petitioner stated in the videos that she read on the Internet about dystonia and learned that some dystonia patients could run and not walk. Then she went and experimented and, each time, walking backward

worked, which Dr. Lancaster attributed to the power of suggestion coming from the information on the Internet. Id. at 633.

Dr. Lancaster cogently explained that petitioner's manifestations of various symptoms involving numerous anatomic sites is exactly the behavior the Hallett article on psychogenic movement disorders describes (Ex. ZZ). As petitioner's treating neurologist Dr. Stephenson depicted in his notes of December 2009, petitioner's movements had variability (manifesting different planes of direction), suggestibility, distractibility, and instant appearance and disappearance at multiple loci. Med. recs. Ex. 22, at 36-37.

What Dr. Steinman could not explain medically was why Dr. Buttar's treatment of petitioner with IVs of saline and/or glucose in water would not only cure her POTS, but also her inability to walk and speak appropriately. Dr. Steinman attributed that "cure" to the placebo effect. But he adamantly continued to believe that petitioner's alleged dystonia was real. The undersigned finds Dr. Lancaster's opinion that petitioner had astasia-abasia, i.e., conversion disorder, more persuasive than Dr. Steinman's opinion that petitioner had dystonia. Dr. Lancaster said he has had 50-100 patients seeing him for a neurological diagnosis who, in fact, manifested conversion disorder. Dr. Steinman never testified that he has the experience of patients who seek his neurologic help but in fact had conversion disorder. Dr. Lancaster, through his greater experience of conversion disorder, is the more credible expert on this diagnosis than Dr. Steinman.

In addition, although Dr. Steinman emphasizes the importance of "boots on the ground" for those doctors having opinions favorable to petitioner's allegations, he ignores the fact that most of these doctors did not see the videos that Dr. Lancaster, Dr. Steinman, and the undersigned saw. Petitioner asserts that Dr. Gee saw the videos, and she asserted that he thought they bore out that she had dystonia. But when the undersigned ordered petitioner to obtain and file a statement from Dr. Gee about his opinion, according to petitioner's attorney in a status report, Dr. Gee never gave her a statement. Moreover, when petitioner was transported by EMTs to Medstar Georgetown Hospital on the second day of the hearing, staying there for a few days, Dr. Gee phoned Dr. Barry and told him petitioner had a "non-organic dystonia to a flu shot." Med. recs. Ex. 179, at 6. Dr. Steinman seems to have ignored Dr. Gee, an important set of boots on the ground, who is currently acting as petitioner's treating neurologist and recognizes that there is not an organic dystonic reaction to a flu vaccination and transmits that information to another treating neurologist, Dr. Barry, who wrote it in the Georgetown Hospital records. Petitioner's reputation at Georgetown Hospital must have been extensive for the dietician Kelsie N. Hitesman to put in her own notes "psychogenic stridor." Med. recs. Ex. 183, at 90.

Dr. Steinman's reliance on boots on the ground is misplaced when the doctors upon whose opinion he relies (including Dr. Sheean) were never privileged to watch the videos of petitioner in October 2009. Moreover, the doctors who examined petitioner even without the assistance of videos expressed numerous concerns over whether her symptoms were organic:

1. Inova Fairfax Hospital ED on September 27, 2009, just one month after vaccination, had the first treaters to diagnose petitioner with conversion disorder. Dr. Jonathan Bresner, a neurologist, noted petitioner's "movements are extremely peculiar and not easily described from a neurological standpoint. It is also odd that the patient's symptoms resolved for a long enough period each morning for her to apply makeup but then return so forcefully that she is unable to speak or move her limbs in any sort

of coordinated motion. The patient is able to walk despite her inability to control her limbs." Her neurological examination was otherwise normal and she had no signs or symptoms of GBS. A psychogenic etiology remained a consideration and she might benefit from psychological counseling or a psychiatric evaluation. Med. recs. Ex. 44, at 31-32.

2. Dr. Mohammamed Mannan wrote on September 29, 2009 that petitioner's condition had a possible psychogenic etiology. Petitioner had very odd neurological symptoms. Med. recs. Ex. 44, at 13. Dr. Mannan noted the discrepancy of petitioner putting on makeup including eyeliner in the hospital, but unable to hold a cup of water in her hand. When the nurses confronted her, she stopped putting on makeup. Petitioner's PCP Dr. Rodriguez contacted Dr. Mannan and said he did not see any organic etiology either, he was concerned about a psychogenic etiology, and he agreed that petitioner should have a psychiatric evaluation. Id. at 14.
3. Dr. Anjail Sharrief at Johns Hopkins Hospital on October 3, 2009 wrote petitioner's neurological examination had many components that were not physiologic, and likely had a psychological overlay. Med. recs. Ex. 2, at 14.
4. Dr. Victor C. Urrutia, a neurologist, on October 3, 2009 wrote petitioner had a strong psychogenic component to her symptoms and she did not have GBS. Med. recs. Ex. 55, at 12. A neurologic progress note identified petitioner's gait as astasia-abasia. Id. at 85. Dr. Urrutia also wrote that petitioner's symptoms "do not fit a physiologic paradigm" and could be due to anxiety. Id. at 15. Dr. Urrutia suggested petitioner see a therapist and noted she felt better after taking Ativan. Id.
5. Dr. Christopher Oakley on October 4, 2009 wrote petitioner was having a stress reaction and anxiety. He recommended she see a therapist or a psychiatrist. Med. recs. Ex. 55, at 6.
6. Dr. Garry Ho wrote on October 6, 2009 that if petitioner did not have anything further on work up, he would strongly consider she had a conversion disorder, delusional disorder, or other psychogenic etiology. Med. recs. Ex. 22, at 14. He also notes that petitioner said she was perfectly normal for 10 hours when she took her then-husband's valium. Id. at 12.
7. Dr. Randolph R. Stephenson, a neurologist, wrote on December 7, 2009 that petitioner had astasia-abasia when he saw her gait on physical examination. Med. recs. Ex. 22, at 37. He noted she had a very clear functional component to her physical examination and no neurological findings that would suggest any particular organic disease. Id.
8. Dr. Reuben Cintron, a neurologist, wrote on January 19, 2010 that petitioner had "some bizarre aspects to her disease." Med. recs. Ex. 1, at 9. He also noted on March 16, 2010 that petitioner's symptoms were still bizarre and there was no objective evidence of where the problems were coming from. Id. at 7. He added on February 3, 2011, "In summary her medical and neurological situation has been at best confusing and very difficult to intellectually define." Med. recs. Ex. 16, at 1. He stated petitioner was exercising "almost to a pathological amount." Id.
9. Dr. Frisca Yan-Go, a neurologist specializing in autonomic neuropathy, wrote on August 15, 2010 that petitioner had a functional overlay and conversion reaction. Med. recs. Ex. 18, at 1.

10. Dr. Daniel Wilkinson, a cardiologist, wrote on February 2, 2011 that petitioner had multiple "very unusual symptoms." Med. recs. Ex. 12, at 1.
11. Dr. Patrick Lyden, a neurologist, wrote on January 10, 2012, that petitioner had a "very complicated case" and an "unsuspected psychiatric diagnosis." Med. recs. Ex. 54, at 10. He added on January 13, 2012 that petitioner "embellished symptoms" and needed a good psychological evaluation. Id. at 12.
12. Dr. Michael Olek, an osteopath, wrote on February 29, 2012, "At this time, it is difficult to have a unifying diagnosis." Med. recs. Ex. 61, at 4.
13. Dr. Joey Gee, an osteopath functioning as petitioner's neurologist, wrote her underlying diagnosis was an "enigma." Med. recs. Ex. 115, at 5. Dr. Gee noted on July 18, 2012 that petitioner's neurologic tests were unremarkable. Id. at 10.
14. Dr. Brian Barry, a neurologist, wrote on June 15, 2016 that petitioner had "effort dependence" on physical examination at Medstar Georgetown University Hospital. Med. recs. Ex. 179, at 5. On that same date, Dr. Gee telephoned Dr. Barry and told Dr. Barry that petitioner had a "non-organic dystonia attributed to a flu shot." Id. at 6.

The negative opinions of the above 14 doctors about the medical legitimacy of petitioner's complaints were from her physical examinations, yet Dr. Steinman ignores their concerns even though their boots were on the ground at the time petitioner claimed the illness that Dr. Steinman now interprets as dystonia.

Dr. Steinman also ignores petitioner's misinforming doctors by giving them a history that she had GBS when her doctors on more than one occasion in two separate hospitals examined her specifically looking for GBS and concluded that since she had normal reflexes, a normal protein count in her CSF, and no paralysis, she did not have GBS. When doctors heard from her that she had GBS, they were more likely to ascribe her history of having autonomic nervous system symptoms to the well-established component of a reaction to flu vaccine. On the witness stand, Dr. Steinman agreed that autoimmune autonomic neuropathy and GBS are at either end of the spectrum of inflammatory neuropathy and did not press his written view that autoimmune autonomic neuropathy was a variant of GBS. Moreover, he finally admitted in his testimony that GBS was out of the case.

Dr. Steinman also ignores petitioner's misinforming doctors by giving them a history that she had optic neuritis when Dr. Swaraj Bose, a neuro-ophthalmologist with stellar credentials, examined her and found nothing wrong with her eyes. Petitioner's website (for which the undersigned would give the URL but the undersigned redacted this case to petitioner's initials at petitioner's request) shows her using a straight edge of a paper in order to read a computer screen, as if she had something wrong with her eyes. Having vision problems is one sign of myasthenia gravis which Dr. Sheean diagnosed petitioner as having. Dr. Sheean is the doctor upon whom Dr. Steinman relies in opining that petitioner has myasthenia gravis, even though it is not Dr. Steinman's "favorite" diagnosis. He said he was not personally wild about that diagnosis and myasthenia gravis was not a cornerstone of his opinion.

Finally, Dr. Steinman diagnosed petitioner with only autoimmune autonomic neuropathy and vagus nerve injury. Petitioner's misinforming doctors about her history did not persuade him even a little that petitioner has conversion disorder. When pressed at the hearing whether petitioner could have both conversion disorder and physical illness, he mused that one day we

may find an organic reason for psychogenic behavior. He said a person could have an illness that was so upsetting that it led to abnormal behavior. Yet, he had just testified Dr. Buttar's use of repeated IVs on petitioner worked not only for her POTS by hydrating her, but also improved her alleged inability to walk forward and speak normally because they had the placebo effect. He recognized IVs cannot improve someone's ability to walk forward and speak normally because blood volume is not pertinent to those activities.

The impression the undersigned received from Dr. Steinman's testimony as well as from his expert reports is he is uncomfortable staying with one position and flits back and forth from opinion to opinion to assist petitioner in prevailing, even if he does not eventually believe what he wrote or said. Dr. Steinman is the essence of a team player, in other words, an advocate. Dr. Steinman's testimony is inconsistent with his own opinions on the stand as well as his opinions in his expert reports, inconsistencies that do not engender credibility.

Dr. Steinman on the witness stand finally rethought and narrowed down what his opinion was: petitioner had autoimmune autonomic neuropathy and vagus nerve damage that her August 23, 2009 flu vaccination caused. As Dr. Lancaster critically notes, the events depicted in the videos and the initial physical examinations of petitioner occurred within the first couple of months after petitioner's August 23, 2009 flu vaccination. If petitioner later had other illnesses, such as POTS, gastroparesis, and/or myasthenia gravis, petitioner has not alleged that flu vaccine caused these diseases months if not years later. The first few months after the flu vaccination, from August 23 to October 23, 2009, make or break petitioner's chance of prevailing in this case, a point Dr. Lancaster made.

Dr. Steinman tried repeatedly in his 10 reports to create a grand picture: flu vaccine caused her rhabdomyolysis, GBS, and myasthenia gravis, whose onsets occurred at the same time (20 days post-vaccination). Then, he moved on to dystonia and to dysautonomia. Then he progressed to autoimmune autonomic neuropathy/autoimmune autonomic ganglionopathy. He was still hanging on to his "inference" of GBS. Further down the road, he added gastroparesis and POTS. Finally, he decided she had anti-GAD antibody syndrome which was undetected earlier because no one tested petitioner's GAD antibodies in 2009. Respondent through experts and hardworking counsel hammered away at the grand picture until only autoimmune autonomic neuropathy and vagus nerve damage were left.

After petitioner slumped to the ground growling during Dr. Steinman's testimony on the second day of the hearing, the undersigned asked Dr. Steinman to attend to her until the EMTs arrived. When the EMTs removed petitioner from the hearing room to the ambulance, Dr. Steinman returned to the witness stand and all his usual conviviality went out of him. He looked disturbed and disheartened. He kept repeating the same statement in response to the undersigned's question--that petitioner was moving air, i.e., breathing, and she was not cyanotic. Respondent's counsel Ms. Walters sensed Dr. Steinman's disquietude as well and asked on cross-examination whether he actually believed petitioner had myasthenia gravis to which he responded in the affirmative. But Ms. Walters whittled Dr. Steinman down to what he really believed and he said the illnesses she has due to the vaccine were autoimmune autonomic neuropathy and vagus nerve damage. He did not mention myasthenia gravis.

His disinclination for that diagnosis surfaced again in the unusual caveat in the Amended Petition that petitioner filed after the hearing that myasthenia gravis was not Dr. Steinman's "favorite" diagnosis but he relied on Dr. Sheean to support it (and petitioner was still alleging flu

vaccine caused it). Yet, Dr. Steinman testified that he did not believe petitioner has Lambert-Eaton Syndrome, another one of Dr. Sheean's diagnoses. Clearly, the fact that Dr. Sheean diagnoses an illness is not determinative of Dr. Steinman's agreement with Dr. Sheean. But petitioner did not allege flu vaccine caused Lambert-Eaton syndrome and thus Dr. Steinman did not need to comment on it. The firefly appearance and disappearance of myasthenia gravis as a diagnosis Dr. Steinman accepted reduced his credibility.

Dr. Steinman asked in one of his expert reports what else could have triggered petitioner's various conditions if not the flu vaccine (ignoring the presence of a viral infection because he claims no one knows the identity of the virus and therefore it does not count as a cause). The Federal Circuit in Grant states petitioner's burden is to prove vaccine causation with affirmative evidence. 956 F.2d at 1149. Saying since there is no other cause, it has to be the vaccine is not affirmative proof.

Dr. Lancaster explains the reason for petitioner's conversion disorder is a subconscious manifestation of stress. A number of petitioner's treating doctors attribute her symptoms to stress or anxiety (Dr. Urrutia, Dr. Oakley) and prescribed benzodiazepines to alleviate her stress or anxiety. They also recommended she get psychological or psychiatric help (Dr. Bresner, Dr. Rodriguez, Dr. Urrutia, Dr. Oakley, Dr. Lyden).

Conversion disorder gives the person who manifests it attention as well as relief from anxiety. See supra, n.27. Dorland's lists the reasons for conversion disorder as: (1) exacerbation of symptoms at times of psychological stress, (2) relief from tension or inner conflicts (primary gains) provided by the symptoms, or (3) secondary gains (support, attention, avoidance of unpleasant responsibilities) provided by the symptoms.

Video Evidence in Support of Conversion Disorder

During one of petitioner's many conversations with Dr. Buttar that video captured, in one,[166] dated October 21, 2009, she and Dr. Buttar exclaim that divine intervention brought them together and that her gut was right: mercury was the cause of her condition. She tells Dr. Buttar he is her hero. Dr. Buttar tells her she is his hero. Dr. Buttar says thank God for the flu shot so we could learn all this. He says they were opening the pathways that day. He says petitioner needed an IV[167] in case her illness comes back. Petitioner says she is "so waiting for Blue Cross Blue Shield's rejection letter" of her claim for reimbursement. Dr. Buttar says petitioner needs oxygen. She says she needs deep brain stimulation and that she has pure dystonia. Dr. Buttar says this is going to be big, and health care in general is going to change. Petitioner says her insomnia is gone. She says she can feel stuff moving back up into her head. She states mercury went back into her head and paralyzed her tongue, neck, and face, giving her cold spots. Dr. Buttar says this proves she does not have a psychogenic component to her condition. Petitioner says it will be hard to make a movie out of her life and wonders who would play her in the movie and portray the physiologic facts. Dr. Buttar says she is having petit mal seizures and acute spasms. He says there is probably enough evidence of that without her having an EEG.

[166] This dialogue starts at minute 7:18 on Ex. 58, Video #409_0514_07. This is part of a terabyte of videos that petitioner filed as Exhibit 58 on a hard drive on May 9, 2012.

[167] Dr. Buttar is not recommending IVIG, but a standard IV. Ex. 58, Video #409_0299_01, at minutes 4:50–5:48, in a phone conversation Stan Kurtz (onscreen) has with Dr. Buttar (offscreen), Dr. Buttar endorses IV fluids but not IVIG which he thought would be detrimental to petitioner and could hurt her.

In another video,[168] dated October 20, 2009, petitioner walks forward without problem to her then-husband in Dr. Buttar's clinic waiting room, puts her arms around her then-husband, tells him she feels good and that she can walk backward, forward, and sideways, exclaims this is great, and kisses her then-husband. She tells Stan Kurtz's wife Michelle on a cellphone that "they fixed her." She says she can walk and talk. She says it is huge. She says she will show Michelle all her new tricks. She states, "It went away so fast too." She says it was the same burning sensations she had in the MRI at Johns Hopkins because MRIs drew metals to the back of her brain. She says she knew it had to be metal because nothing else could pull the mercury down. Petitioner talks about being free again and expresses sympathy for poor autistic kids. She says she does not know how they deal with it, presumably autism. She says Johns Hopkins diagnosed her with dystonia and they cannot back out of it. But they said dystonia has no cure; she would be paralyzed for life. They said she had an irreversible reaction to the flu shot. (None of this is in the Johns Hopkins medical notes. Only a physical therapist there said petitioner had dystonia.)

In another video,[169] dated October 21, 2009, Stan Kurtz is having a conversation with Michael, one of Dr. Buttar's employees. Stan Kurtz says many people told him not to come. A friend of his who is a neurologist who had looked at the videos of petitioner warned him that petitioner has a psychogenic disorder. His neurologist friend said he was 99.9 percent sure it was psychogenic. He said Stan Kurtz should not put his reputation and the reputation of Generation Rescue on the line by associating with petitioner. He said he was telling Stan Kurtz this as a friend. When Stan Kurtz said petitioner's heart rate was 120 beats per minute, his friend said that was an additional condition to the psychogenic disorder.

Stan Kurtz also told Michael that the former head of the National Institutes of Health, Bernadine Healy,[170] was a friend. She had been mourning the death of her mother when Stan Kurtz telephoned her. She warned him to stay away from petitioner and not to put Generation Rescue's reputation at risk. She said petitioner had an eating disorder, perfect makeup, and was sitting up. She said Stan Kurtz should get an EEG done to prove petitioner's condition was not psychogenic. Stan Kurtz said to Michael that people will marginalize petitioner and say that she has a rare strange condition. But Stan Kurtz said he would have evidence, get the Johns Hopkins records, and let the facts speak for themselves.

The undersigned finds quite remarkable that Stan Kurtz had two friends, a neurologist and the noted Dr. Bernadine Healy, separately warn him away from associating with petitioner after seeing the videos of petitioner on national television. Both separately told Stan Gertz that petitioner has a psychogenic disorder and that if he continued associating with her, he risked his own and Generation Rescue's reputations.

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[168] The pertinent parts are between minutes 1-5 on Ex. 58, Video #409_0438_01.

[169] The pertinent parts are between minutes 6-13 on Ex. 58, Video #409_0474_02.

[170] Bernadine Healy was the 13th director of NIH, and the first woman to head NIH. She died from brain cancer at the age of 67 on August 6, 2011. "The Age of Autism anti-vaccine group named her 2008 Person of the Year for her support of the discredited hypothesis that vaccines are linked to autism." Bernadine Healy, WIKIPEDIA, THE FREE ENCYCLOPEDIA , https://en.wikipedia.org/wiki/Bernadine_Healy (last visited April 5, 2019). Dr. Healy gave an interview on CBS News on May 12, 2008 that the existing vaccine court claims prove vaccines can cause harm. Leading Dr.: Vaccines-Autism Worth Study, CBS NEWS (May 12, 2008), https://www.cbsnews.com/news/leading-dr-vaccines-autism-worth-study/.

Ironically, Dr. Buttar and petitioner had an agreement that she would be the proponent of his IV and hyperbaric oxygen therapy treatments on the show 20/20, but she broke her agreement with him and said on national television that those treatments had not cured her. By rejecting the "cure" on national television, petitioner continued from 2009 through 2016 with more symptoms, more doctors, and more drugs, and onto IVIG therapy. She also ended up paying for her "treatment" with Dr. Buttar because Stan Kurtz refused to pay for it.

Dr. Steinman and Dr. Lancaster were total opposites in their opinions about whether petitioner has a lasting physical illness and on the value of the videos filed into evidence. Dr. Steinman dismissed the videos as less important than meeting petitioner in person right before the hearing rather than watching, as he put it, a "little screen," although he did testify that watching the videos convinced him petitioner had dystonia. Dr. Lancaster, on the other hand, valued the worth of the videos in giving him an opportunity to observe petitioner's behavior directly over many hours. He testified that he was able to stop, go back, view something again, look at it in detail, and observe petitioner's gait, speech, and other symptoms. Dr. Lancaster said that with these videos, he had been able to study petitioner and diagnose her with conversion disorder with the most detail of any patient he has had.

What a vast difference in approach in these two neurologic experts. Dr. Steinman was cavalier in dismissing the value of the videos, except for saying they confirmed his opinion petitioner had dystonia. He testified he did not think petitioner had dystonia until he saw petitioner's weird movements going away when she walked backward in the videos. Tr. at 492-93. Dr. Steinman questioned how could anyone know that walking backward would make her flailing go away. Id. at 493. But Dr. Lancaster pointed out that petitioner stated in the videos that she read on the Internet about dystonia and learned that some dystonia patients could run and not walk. Then she went and experimented and, each time, it worked. Id. at 633. Dr. Lancaster stated this was an example of suggestion which worked for petitioner because she thought it would.

The undersigned views Dr. Lancaster as the more persuasive expert in this case. Both experts agreed on one point however: the powerful placebo effect of Dr. Buttar's suggestion to petitioner that she would get better to which petitioner had a great response.

The undersigned finds that more likely than not petitioner has conversion disorder.

Does petitioner have any physical illness that flu vaccine caused?

Dr. Steinman testified that someone can have both conversion disorder and a physical illness. He agreed that Dr. Buttar's treatment had a placebo effect on petitioner because not only did the IV (without immunoglobulin) "cure" her POTS, it also "cured" her alleged inability to walk forward and to speak normally, at least for a time. This leads to the question whether petitioner had any physical illness other than rhabdomyolysis, recognizing as Dr. Steinman did that petitioner's serious medications could have caused her gastroparesis, as Dr. Lancaster said.

Dr. Steinman constantly shifted in his 10 expert reports about what he thought happened in this case. Dr. Steinman testified that the case is complex, rare, and difficult, which is true. The undersigned assumes the many demands on Dr. Steinman's time and the numerous cases in which he is petitioners' expert have made him incapable of focusing until he gets to the hearing because he mentioned several times at the hearing that "now" that he was at the hearing, he could

focus on the issues. His shape-shifting necessitated an extraordinary amount of effort not only on the parts of respondent's experts, Dr. Lancaster and Dr. Whitton, to respond to Dr. Steinman's ever new theories, but also on the part of the undersigned. So many statements he made at the hearing, such as he has no proof of any component of flu vaccine that is homologous to petitioner in molecular mimicry explanation of her autoimmune autonomic neuropathy, set everyone else in catch-up mode. GBS dropped out of the picture although he had focused on it as the explanation of everything in several of his reports. Myasthenia dropped out of the picture when he realized he did not believe petitioner had it. Anti-GAD65 antibody syndrome, stiff person syndrome, and intestinal pseudo-obstruction dropped out of the picture when he never testified about them.

When discovery is not the norm and expert reports replace depositions as in the Vaccine Program, an expert who can never make up his mind is practically useless. A summary of Dr. Steinman's metamorphoses follows.

Dr. Steinman's first expert report (Ex. 65) was that flu vaccine caused petitioner's rhabdomyolysis followed by serious autoimmune dysautonomia, based on a theory of inflammation directed to myelin in portions of the autonomic nervous system via molecular mimicry. He also states that tests of petitioner's sera indicated she had lupus as well as dysautonomia. He focuses on how flu vaccine causes GBS. He states petitioner had autoimmune ganglionopathy.

Respondent's initial neurologic expert Dr. Donofrio focuses on the variability on distraction of petitioner's symptoms. Ex. B. Dr. Donofrio states Dr. Steinman ignored all the anomalies in petitioner's presentation and did not explain how they are consistent with his diagnosis. Testing did not show petitioner having a significant change in pulse or blood pressure during up-tilt testing to substantiate the diagnosis of either POTS or orthostatic hypotension. He states petitioner did not have symptoms that accompany autoimmune ganglionopathy. He denies petitioner had GBS because she had normal reflexes and normal strength. Dr. Donofrio thinks petitioner's viral illness (which Dr. Steinman ignores) is the more likely cause of her rhabdomyolysis.

Dr. Steinman's first supplemental expert report (Ex. 92) clarifies that flu vaccine caused both petitioner's rhabdomyolysis and her autonomic autoimmune neuropathy, but that rhabdomyolysis did not cause her autonomic autoimmune neuropathy. He recognizes that petitioner will not receive compensation under the Vaccine Program for rhabdomyolysis because she did not have sequelae of it for more than six months.

Dr. Steinman's second supplemental expert report (Ex. 108) states that petitioner had a variant of GBS which no one recognized and that two treaters (one of whom was at the tertiary care center Johns Hopkins) denied. Dr. Steinman's theory for the treaters' denying petitioner had GBS was that she did not have classic GBS, i.e., she did not have signs or symptoms of GBS (areflexia, paralysis, increased protein in her cerebrospinal fluid). He posited that pure dystonia can be a GBS variant, citing three pieces of medical literature, none of which supports that view. Finally, on the witness stand, he admitted that autoimmune autonomic neuropathy is at one end of the spectrum of inflammatory neuropathy and GBS is at the other end of the spectrum of inflammatory neuropathy. He admits that he infers petitioner had GBS since she had autonomic dysfunction. Finally, he dispenses at the hearing with GBS altogether.

Respondent's second neurologic expert Dr. Lancaster gives his opinions about the videos showing petitioner flailing her arm, bopping her head, while walking forward in astasia-abasia gait. Ex. H. He states that petitioner's viral infection and racing training were the more likely cause of her rhabdomyolysis than flu vaccine. He notes that rhabdomyolysis does not damage the autonomic nervous system or the central nervous system. His summary of the medical records forms the basis of his opinion that petitioner has conversion disorder.

Respondent's virologic and immunologic expert Dr. Whitton points out that when petitioner had rhabdomyolysis, she did not have any neurologic complaints. Ex. Z. He disagrees with Dr. Steinman's use of molecular mimicry in this case because humans have only about 20 different amino acids and it is inevitable a scientist will find identical sequences and multiple homologies. This response from Dr. Whitton is obviously based on Dr. Steinman's initial claim that he found homology, which he denied at the hearing.

Dr. Steinman's third supplemental expert report (Ex. 118) states petitioner had three illnesses, all beginning at the same time (20 days after vaccination) and all with the same symptoms of fatigue and weakness. The three illnesses are rhabdomyolysis, autonomic autoimmune neuropathy, and myasthenia gravis. He disputes Dr. Lancaster's opinion that petitioner has conversion disorder on the basis that Dr. Lancaster has not met her or treated her. Dr. Steinman ignores the contemporaneous videos that form the basis of Dr. Lancaster's opinion.

Respondent filed Dr. Lancaster's first supplemental expert report (Ex. RR), noting that Dr. Sheean's diagnosis of petitioner having myasthenia gravis was likely incorrect. Dr. Lancaster states that dystonia is not a symptom of autoimmune autonomic neuropathy because dystonia comes from the brain and not from the autonomic nervous system.

Respondent filed Dr. Whitton's first supplemental expert report (Ex. SS), stating Dr. Steinman's theories are jumbled.

Dr. Steinman's fourth supplemental expert report (Ex. 141) still discusses GBS as if petitioner had it. He then comments on the contemporaneous videos. He refuses to comment on petitioner's singularly narrow attempt to walk forward with flailing arms. As for her bobbing her head violently, he states repeatedly that he would need a concomitant EEG and video monitoring to know if this was an actual seizure. He ignores petitioner's normal prior EEGs.

After the hearing, petitioner filed Dr. Steinman's fifth supplemental expert report (Ex. 181) and respondent filed Dr. Whitton's second supplemental report (Ex. YYY), stating the same opinion that petitioner's negative testing for flu A and B virus antigen on September 12, 2009 did not mean flu vaccine was ineffective.

Dr. Steinman's sixth supplemental expert report (Ex. 191) states that petitioner at Medstar Georgetown University Hospital was noted to have ptosis which confirms she has myasthenia gravis. He fails to comment on the treating neurologist Dr. Barry's statement that petitioner's ptosis could be overcome with upward gaze, which means petitioner did not have true ptosis. Dr. Steinman focuses on anti-GAD antibodies as proof that petitioner had an autoimmune reaction to flu vaccine, although she was not tested for anti-GAD antibodies in 2009.

Dr. Whitton's third supplemental expert report (Ex. ZZZ) states Dr. Steinman's previous analogy of homologies between flu vaccine and petitioner's various conditions relied upon Dr.

Steinman's error in picking 2010-2011 flu vaccine components instead of 2009-2010 flu vaccine components. Thus, Dr. Steinman switched from homology to myelin basic protein to homology to myelin oligodendroglial glycoprotein and 2,3 CNPase. Dr. Steinman also invoked another protein, i.e., GAD. Dr. Steinman seemed to be favoring a stiff person syndrome diagnosis. Dr. Whitton attributed petitioner's elevated GAD antibodies to her receiving IVIG therapy which contained anti-GAD antibodies from the donors.

Dr. Lancaster's second supplemental expert report (Ex. FFFF) says stress may have triggered petitioner's stridor during the second day of the hearing. Dr. Lancaster states petitioner does not have stiff person syndrome. He notes petitioner's ptosis at Georgetown could be overcome by looking up, which means it was psychogenic. He also attributed petitioner's elevated GAD antibodies to IVIG infusions and wrote petitioner did not have any syndromes associated with GAD65 antibodies. Dr. Lancaster states it would be entirely unprecedented for one patient to have so many presumably autoimmune disorders affecting so many distinct areas of her nervous system, and even more remarkable, that her condition remits completely, sometimes in seconds, leaving no convincing objective evidence of its existence.

Dr. Steinman's seventh supplemental expert report (Ex. 198) states petitioner's collapse and stridor in the hearing room the second day of the hearing proves she has myasthenia gravis. He insists her anti-GAD antibody test results prove she has an ongoing immunologic process. He says the anti-GAD antibodies explain her having myasthenia gravis, stiff person syndrome, intestinal pseudo-obstruction, and autonomic neuropathy.

Dr. Steinman's eight supplemental expert report (Ex. 206) says all of petitioner's problems are related to anti-GAD antibodies.

Dr. Steinman's ninth supplemental expert report (Ex. 208) says flu vaccine likely triggered petitioner's autoimmunity to GAD.

Dr. Whitton's fourth supplemental report (Ex. MMMM) says petitioner had much lower anti-GAD antibodies than someone with stiff person syndrome would have. He disagrees with Dr. Steinman's thesis that homology proves molecular mimicry occurred. Dr. Whitton states proteins fold into complex three-dimensional structures and the relevant part of the host protein may be hidden inside the fold and therefore inaccessible to antibodies.

Dr. Lancaster's third supplemental report (Ex. QQQQ) states that none of the disorders Dr. Steinman says petitioner has (myasthenia gravis, autonomic failure, and dystonia) accounts for petitioner's most prominent symptoms in the weeks and months after her flu vaccination.

Petitioner had a physical illness rhabdomyolysis whose cause could be the viral infection that Dr. Steinman ignores because he does not know what kind of virus petitioner had in early September 2009. In addition, petitioner was training for a race and exercise can provoke rhabdomyolysis, particularly in someone who was not yet over her upper respiratory infection. As for vagus nerve damage, petitioner has been diagnosed with orthostatic hypotension due to volume depletion, i.e., she was not drinking enough fluids, causing her to faint. This has nothing to do with flu vaccination. Her doctors note this connection of vasovagal syncope with dehydration, and also note she was not orthostatic when hydrated:

1. Dr. Jeffrey Luy, a cardiologist, wrote on September 18, 2009 that petitioner's syncope was "probably vasovagal due to some relative element of dehydration." Med. recs. Ex. 51, at 25.
2. Dr. Mannan, attending physician in Inova Fairfax Hospital, wrote on September 29, 2009 that she had orthostatic hypotension due to volume depletion. Med. recs. Ex. 44, at 13. Her hypotension improved with IV hydration. Id.
3. Dr. Farhad Zangeneh, an endocrinologist, wrote on May 24, 2010 that petitioner's blood pressure when she was sitting and standing was nearly identical. He wrote she did not have orthostatic hypotension. Med. recs. Ex. 43, at 29.
4. Dr. Yan-Go, a neurologist specializing in autonomic neuropathy, wrote on August 24, 2010 that petitioner was not orthostatic. Med. recs. Ex. 18, at 3. She also wrote on September 14, 2010 that petitioner did not have serious pure autonomic failure or degenerative dysautonomia. Id. at 6.
5. Dr. Wilkinson, a cardiologist, wrote on February 3, 2011 that petitioner was taking very potent medications and did not have unusual syncope. Med. recs. Ex. 12, at 1.

Dr. Steinman and Dr. Lancaster agreed on six medical points: (1) that a technician's interpretation of a SPECT scan to show that petitioner had abnormal cerebral perfusion was wrong; (2) that petitioner's drugs (particularly Sandostatin) may have caused her gastroparesis; (3) that Dr. Buttar's eight-hour IV infusions of water and glucose/saline had a placebo effect in "curing" albeit temporarily petitioner's alleged POTS, alleged dystonia, and alleged foreign accent syndrome; (4) that there is no such diagnostic entity as "Raynaud's of the brain," (5) that hyperbaric oxygen therapy and chelation have only limited utility and no utility whatsoever for petitioner's complaints; and (6) that petitioner does not have Lambert-Eaton Syndrome (one of the diagnoses Dr. Sheean posited as a diagnosis for petitioner). That petitioner in early September 2009 had rhabdomyolysis is not an issue. It is also not an issue that petitioner recovered quickly from rhabdomyolysis.

As is obvious from the description of Dr. Steinman's constantly changing analysis of petitioner's purported illnesses, he never had a grasp of this case and that is not only because the case is complex. He never focused on it, he brushed away the many indications of conversion disorder and, finally, at hearing, admitted on cross-examination that now that he was focused on the case, his only diagnoses were autoimmune autonomic neuropathy and vagus nerve damage. He flitted through 10 expert reports with constant changes of his opinion. Then, in his testimony, he changed his mind again.

Dr. Steinman's frequent carping at Dr. Whitton that he is not licensed to practice medicine in the United States is irrelevant since Dr. Whitton is a researcher, not a clinician. Dr. Whitton's purpose at the hearing was to counter Dr. Steinman's age-old attempt to shoehorn molecular mimicry into every causation in fact case. As for lack of board certification, although Dr. Steinman is not board certified in immunology, he testifies not only as an expert neurologist (and he is board certified in neurology) but also as an expert immunologist. Respondent did not object to his expertise in immunology. Dr. Sheean, for whom Dr. Steinman provided copious encomiums, is not board certified in anything.

Dr. Steinman denied at the beginning of his testimony that he is an advocate and that he would not delve into the law. He pictured himself as just a neutral physician whose purpose was to educate the special master about the significance of the medical facts of the case. Would that

were so. Dr. Steinman is indeed an advocate. He cited legal decisions in more than one of his expert reports. He cherry picked among the evidence to support the conclusion he wanted the special master to reach. But he never completely focused on the issues until the hearing when his numerous other vaccine cases and professional chores were not before him. He even mistook the date of the flu season he was checking to note the components of the 2009 flu vaccine petitioner received as part of his analysis of molecular mimicry, picking the 2010-2011 flu season instead of the 2009-2010 flu season. That made numerous reports he wrote erroneous.

Dr. Steinman had a valid concern about participating in this case because of the presence of Dr. Buttar and petitioner's participation in numerous public media. The undersigned can appreciate Dr. Steinman's concern. He has an extraordinary list of accomplishments in his CV. But accomplishments are not enough when he squandered his own time and that of respondent's experts, both counsel, and the special master by pulling out various diagnoses and analyses like rabbits from a hat without grasping the facts of this complex case.

It is problematic when an expert witness is at war with himself, proposing diagnoses and then negating them, eventually ending up with pure speculation that because petitioner had a higher than normal measure of GAD65 in 2015 that GAD associated-antibody disease is the explanation for all her many illnesses going back six years to her 2009 flu vaccination when no one tested her for GAD65 antibodies. Yet, he had bursts of honesty through the haze of his advocacy when he agreed with Dr. Lancaster on the six above-mentioned medical points. He admitted, "We're not saying that it's GBS here, we should make that clear." Tr. at 430.

Consequently, because of Dr. Steinman's many foibles and fumbles, the undersigned found herself relying exclusively on Dr. Lancaster who was methodical, precise, and dedicated to sticking to the record. His opinion and the opinion of Dr. Donofrio are consistent. Dr. Donofrio wrote that petitioner did not have dystonia, dysautonomia, POTS, orthostatic hypotension, or GBS. Ex. B, at 7-8. He believed petitioner probably had a viral illness in early September 2009 which, together with her regimented exercise while training for a race, caused her rhabdomyolysis. Id. at 8.

Dr. Lancaster cogently explained how petitioner's medical records belie her having a physical illness, other than rhabdomyolysis. His opinion is consistent with the opinions of the numerous neurologists petitioner saw at Inova Fairfax, Inova Loudon, and Johns Hopkins. Dr. Lancaster's opinion is consistent with the conclusion of the early treating neurologist, Dr. Stephenson. All these neurologists saw petitioner in the few months after her vaccination and would know best if she were manifesting any neurologic illness. They uniformly denied she had any neurologic illness. Dr. Steinman ignored these records. Dr. Lancaster paid attention to these records, and medically interpreted in great detail what the contemporary videos conveyed. He was a straight arrow from start to finish.

The undersigned also values Dr. Whitton's explanation that Dr. Steinman's reliance on homology to explain how flu vaccine caused whatever Dr. Steinman decided at the moment she had was oversimplistic and does not encompass the complexity of protein folding in three dimensions. It was respondent's expert, not petitioner's expert, who recognized Dr. Steinman initially analyzed the components of the 2010-2011 flu season trivalent flu vaccine, not the 2009-2010 flu season trivalent flu vaccine (the flu vaccine petitioner received was on August 23, 2009), an example of Dr. Steinman's carelessness. The undersigned finds Dr. Whitton's testimony compelling.

What is most perplexing of all the many twists and turns of Dr. Steinman's opinions is his putting dystonia within the category of the autonomic nervous system. Dr. Lancaster persuasively testified that dystonia is not an autonomic nervous system malfunction, but instead is a problem in the central nervous system. Dr. Lancaster said the medical community accepts this understanding of dystonia. Dr. Steinman based his erroneous opinion on four mistakes he made: (1) that petitioner manifested dystonia (Dr. Steinman's supposed "aha" moment when he saw the videos); (2) that dystonia is a problem of the autonomic nervous system; (3) that petitioner had dysautonomia of which dystonia is a part; and (4) that flu vaccine caused all these problems, none of which she had. These mistakes negate Dr. Steinman's credibility.

The word Dr. Steinman kept emphasizing in his testimony as an explanation for his new analysis of the case at the hearing was "focus." In other words, by flying from California to Washington, DC, going to the hotel, meeting petitioner for the first time, and talking with petitioner's counsel, he did not have any distractions from his many other activities. Now he could focus on the case. The undersigned recognizes that Dr. Steinman is a very busy man in his professional life (see supra, nn.121-25 and accompanying text), and that he is or has been petitioner's expert in many vaccine cases.[171] However, his responsibility to petitioner,

[171] This is a listing of decisions published on Westlaw in which Dr. Steinman was petitioner's expert: Abbott ex. rel. Bunch v. Sec'y of HHS, No. 99-497V, 2010 WL 3186269 (Fed. Cl. Spec. Mstr. June 28, 2010); Ricci v. Sec'y of HHS, No. 99-524V, 2011 WL 2260391 (Fed. Cl. Spec. Mstr. May 16, 2011), aff'd, 101 Fed. Cl. 385 (2011); Contreras v. Sec'y of HHS, No. 05-626V, 2012 WL 1441315 (Fed. Cl. Spec. Mstr. Apr. 5, 2012), vacated, 107 Fed. Cl. 280 (2012), 2013 WL 6698382 (Fed. Cl. Spec. Mstr. Nov. 19, 2013), vacated, 116 Fed. Cl. 472 (2014), 2014 WL 8098606 (Fed. Cl. Spec. Mstr. Oct. 24, 2014), aff'd, 121 Fed. Cl. 230 (2015), vacated, 844 F.3d 1363 (Fed. Cir. 2017); Broekelschen v. Sec'y of HHS, No. 07-137V, 2009 WL 440624 (Fed. Cl. Spec. Mstr. Feb. 4, 2009), aff'd, 89 Fed. Cl. 336 (2009), aff'd, 618 F.3d 1339 (Fed. Cir. 2010); Torday v. Sec'y of HHS, No. 07-372V, 2011 WL 2680687 (Fed. Cl. Spec. Mstr. June 20, 2011); Veglia v. Sec'y of HHS, No. 02-397V, 2009 WL 515407 (Fed. Cl. Spec. Mstr. Feb. 10, 2009); Smith v. Sec'y of HHS, No. 08-864V, 2016 WL 2772194 (Fed. Cl. Spec. Mstr. Apr. 18, 2016); McCulloch v. Sec'y of HHS, No. 09-293V, 2015 WL 3640610 (Fed. Cl. Spec. Mstr. May 22, 2015); Brown v. Sec'y of HHS, No. 09-426V, 2011 WL 5029865 (Fed. Cl. Spec. Mstr. Sept. 30, 2011); Tetlock v. Sec'y of HHS, No. 10-56V, 2018 WL 823420 (Fed. Cl. Spec. Mstr. Jan. 19, 2018); Daniel v. Sec'y of HHS, No. 10-745V, 2016 WL 7785955 (Fed. Cl. Spec. Mstr. Dec. 21, 2016); Perez v. Sec'y of HHS, No. 10-659V, 2015 WL 9483680 (Fed. Cl. Spec. Mstr. Dec. 8, 2015); Dillon v. Secy of HHS, No. 10-850V, 2013 WL 33745900 (Fed. Cl. Spec. Mstr. June 25, 2012), aff'd, 114 Fed. Cl. 236 (2014); B.A. v. Sec'y of HHS, No. 11-51V, 2019 WL 460941 (Fed. Cl. Spec. Mastr. Jan. 10, 2019); Musto v. Sec'y of HHS, No. 11-801V, 2017 WL 1150797 (Fed. Cl. Spec. Mstr. Mar. 2, 2017); Vigliotti v. Sec'y of HHS, No. 12-281V, 2019 WL 948365 (Fed. Cl. Spec. Mstr. Jan. 29, 2019); Auch v. Sec'y of HHS, No. 12-673V, 2017 WL 1718783 (Fed. Cl. Spec. Mstr. Apr. 5, 2017); Guerrero v. Sec'y of HHS, 12-689V, 124 Fed. Cl. 153 (2015); Giannetta v. Sec'y of HHS, No. 13-215V, 2017 WL 4249946 (Fed. Cl. Spec. Mstr. Sept. 1, 2017); Taylor v. Sec'y of HHS, No. 13-700V, 2018 WL 6291355 (Fed. Cl. Spec. Mstr. Oct. 30, 2018); Harrington v. Sec'y of HHS, No. 14-43V, 2018 WL 4401976 (Fed. Cl. Spec. Mstr. Aug. 14, 2018); Kukreja v. Sec'y of HHS, No. 14-104V, 136 Fed. Cl. 431 (Dec. 22, 2017); Quance v. Sec'y of HHS, No. 14-271V, 2018 WL 7017750 (Fed. Cl. Spec. Mstr. Dec. 13, 2018); Pentcholov v. Sec'y of HHS, No. 14-414V, 2016 WL 3197389 (Fed. Cl. Spec. Mstr. Apr. 29, 2016); Rolshoven v. Sec'y of HHS, No. 14-439V, 2018 WL 1124737 (Fed. Cl. Spec. Mstr. Jan. 11, 2018); Rosof v. Sec'y of HHS, No. 14-766V, 2017 WL 1649802 (Fed. Cl. Spec. Mstr. Mar. 31, 2017); S.B. v. Sec'y of HHS, No. 14-918V, 2018 WL 6819552 (Fed. Cl. Spec. Mstr. Nov. 28, 2018); Quackenbush-Baker v. Sec'y of HHS, No. 14-1000V, 2018 WL 1704523 (Fed. Cl. Spec. Mstr. Mar. 14, 2018); Forrest v. Sec'y of HHS, No. 14-1046V, 2019 WL 925495 (Fed. Cl. Spec. Mstr. Jan. 28, 2019); D'Tiole v. Sec'y of HHS, No. 15-85V, 2017 WL 5379195 (Fed. Cl. Spec. Mstr. Sept. 19, 2017), aff'd, 132 Fed. Cl. 421 (2017), aff'd,726 Fed. Appx. 809 (Fed. Cir. 2018); Chinea v. Sec'y of HHS, No. 15-95V, 2019 WL 1873322 (Fed. Cl. Spec. Mstr., Mar. 15, 2019), mot. for rev., (Apr. 15, 2019); Crosby v. Sec'y of HHS, No. 15-556V, 2017 WL 7101151 (Fed. Cl. Spec. Mstr. Aug. 29, 2017); Arnold v. Sec'y of HHS, No. 15-534V, 2017 WL 3165486 (Fed. Cl. Spec. Mstr. June 22, 2017); Winterfeld v. Sec'y of HHS, No. 15-933V, 2018 WL 2225178 (Fed. Cl. Spec. Mstr. Mar. 9, 2018 (Fed. Cl. Spec. Mstr. Mar. 9, 2018); Franco v. Sec'y of HHS, No. 16-99V, 2018 WL 4141292 (Fed. Cl. Spec. Mstr. July 31, 2018); Meadows v.

respondent, both counsel, and the undersigned is to give his full attention to and focus on the facts of a case in which he voluntarily agrees to be an expert. To wait until he is actually at the hearing in order to focus is inexcusable. Granted, this is a complex case. But its complexity demands more, not less, of his time and attention. To make respondent, both counsel, and the undersigned careen along the many tracks of his various positions, all of which Dr. Steinman forcefully held until he changed his mind, is unacceptable and lessens any credibility he may have had because of his impressive CV.

When Dr. Steinman was in his pro-myasthenia gravis mode, he supported petitioner's having the disease because a tiny minority of patients have negative results on all the critical tests for myasthenia gravis. But the Vaccine Act, § 300aa-13(a)(1), and the Federal Circuit's interpretation of it in Grant require affirmative proof by preponderant evidence. 956 F.2d at 1149.

The undersigned recognizes that Dr. Steinman abandoned his pro-myasthenia gravis mode when he responded to Ms. Walters' question asking him what precise conditions petitioner had and he answered autoimmune autonomic neuropathy and vagus nerve damage. Even if petitioner did have autoimmune autonomic neuropathy, which considering the evidence in the videos, Dr. Lancaster's interpretation of the videos, and petitioner's negative medical examinations, is seriously in doubt, Dr. Steinman's explanation of how flu vaccine caused petitioner's autoimmune autonomic neuropathy is based on a fallacy.

Dr. Steinman based his causation opinion on a theory that flu vaccine, containing protein homologies to myelin basic protein, resulted in molecular mimicry that caused petitioner autoimmune autonomic neuropathy. Dr. Lancaster said this mechanism does not exist. Attacks on myelin, if they occur, do not select solely the autonomic nervous system. Dr. Lancaster said that he is unaware of evidence showing autoimmune autonomic neuropathy has anything to do with demyelination of the autonomic nervous system. He denied there is such a disease entity as a selective autonomic neuropathy as compared to an autonomic neuropathy that is secondary to and associated with GBS, a demyelinating peripheral neuropathy. (That certainly explains why Dr. Steinman labored so strenuously to prove petitioner had GBS so as to link her putative autonomic neuropathy to it.)

Moreover, Dr. Whitton explained, once respondent's counsel pointed out to Dr. Steinman at the hearing Dr. Steinman's error in selecting the wrong flu season to analyze the components of the flu vaccine that petitioner received, Dr. Steinman's new theory that Brisbane H_3N_2 virus induces a T-cell response has never been proved. In addition, Dr. Whitton testified Brisbane H_3N_2 is not an immune epitope capable of triggering an immune response. The Lei paper (Ex. OOO) showed that humans and mice who received 2009 H_1N_1 vaccine, even though eight of the humans reacted to the vaccine with GBS, did not have antiganglioside antibodies. That vitiates Dr. Steinman's attempt to explain how petitioner's 2009 trivalent flu vaccine caused whatever condition he was attempting to prove by reference to the Nachamkin article (Ex. LLL and Ex. 146) which detected antiganglioside antibodies in mice. This does not of course mean petitioner

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Sec'y of HHS, No. 16-861V, 2018 WL 6292565 (Fed. Cl. Spec. Mstr. Oct. 31, 2018); Zumwalt rel. L.Z. v. Sec'y of HHS, No. 16-994V, 2018 WL 6975184 (Fed. Cl. Spec. Mstr. Nov. 27, 2018); Ahler v. Sec'y of HHS, No. 16-1147V, 2018 WL 2224896 (Fed. Cl. Spec. Mstr. Apr. 9, 2018); Matthaes v. Sec'y of HHS, No. 16-1266V; 2018 WL 4390644 (Fed. Cl. Spec. Mstr. July 30, 2018).

had GBS which is the subject of both the Nachamkin and Lei articles. She did not have GBS. What we are left with is a faulty explanation of how flu vaccine caused a purported autonomic neuropathy that is really conversion disorder.

Except for rhabdomyolysis, viral infection, and possibly gastroparesis, the undersigned finds that petitioner failed to prove she had the diseases she either alleged or were mentioned in the medical records: seizures, GBS, dystonia, dysautonomia, autonomic autoimmune neuropathy/autonomic immune ganglionopathy, neurocardiogenic syncope, vagal nerve damage, optic neuritis, myasthenia gravis, mercury poisoning, MS, lupus, pseudo-intestinal obstruction, poor cerebral perfusion, POTS, anti-GAD antibody syndrome, and stiff person syndrome.

Her viral infection in early September is well established in the medical records with the standard symptoms of nasal congestion, fever, cough, phlegm. Her rhabdomyolysis is well-associated with viral infection and exercise. The two case reports petitioner filed that theorize vaccines may cause rhabdomyolysis are insufficient proof. Crutchfield v. Sec'y of HHS, No. 09-39V, 2014 WL 1665227 (Fed. Cl. Spec. Mstr. Apr. 7, 2014), aff'd, 125 Fed. Cl. 251 (2014). As Dr. Steinman recognized in his reports, petitioner recovered from rhabdomyolysis quickly and would not receive compensation from the Vaccine Program even if flu vaccine played a role in causing it. 42 U.S.C. § 300aa-11(c)(1)(D)(i). The undersigned finds that flu vaccine did not cause petitioner's rhabdomyolysis.

Both Dr. Steinman and Dr. Lancaster thought that petitioner's drugs, particularly Sandostatin, caused her gastroparesis. On August 26, 2010, which is 11 months after petitioner's flu vaccination, Dr. Ghassemi, a gastroenterologist, determined by testing petitioner that she did not have a motility disorder. Med. recs. Ex. 18, at 8-9. The undersigned finds that the drugs petitioner took caused her gastroparesis. Dr. Steinman tried to do an end run around this gap of time between vaccination and gastroparesis diagnosis by saying if petitioner did not have a vaccine injury, she would not have had to take the potent drugs. But the undersigned finds she did not have a vaccine injury and rejects Dr. Steinman's attempt to create a causative link.

The undersigned finds that petitioner had conversion disorder and not a vaccine related injury. Thus, petitioner has failed to make a prima facie case that flu vaccine caused her any condition which she alleged. Regarding an Althen analysis, per the Federal Circuit's holding in Broekelschen, an Althen analysis is unnecessary when petitioner does not have the disease petitioner alleged. See Broekelschen, 618 F.3d at 1346-49.

CONCLUSION

The undersigned **DISMISSES** this case for failure to prove causation in fact.

In the absence of a motion for review filed pursuant to RCFC Appendix B, the clerk of the court is directed to enter judgment herewith.[172]

[172] Pursuant to Vaccine Rule 11(a), entry of judgment can be expedited by each party, either separately or jointly, filing a notice renouncing the right to seek review.

IT IS SO ORDERED.

Dated: May 24, 2019

/s/ Laura D. Millman
Laura D. Millman
Special Master